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A MANUAL OF DIAGNOSIS  
OF THE  
DISEASES OF THE HEART:

PRECEDED BY CLINICAL RESEARCHES,

CALCULATED TO

FACILITATE THE STUDY OF THESE RESEARCHES.

BY

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TRANSLATED FOR THE "WESTERN LANCET,"

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## P R E F A C E.

MUCH has been written on the heart, especially in the course of the few past years. But can this be a reason for ceasing to call public attention to the subject? Perhaps so, if all the problems that attach themselves to this study were at present resolved; if, upon all the principal features of the physiological and clinical history of the heart, a universal concert had replaced the discord which, for so long a time, checked the progress of the science. But it is not thus; we may be even justified in declaring, that at the present moment few organs contribute so much to differences of opinion and multiplied discussions, as the heart. Those who have been willing to undertake this part of our art, and who have even consecrated lengthy researches to it, have encountered doubt and hesitation in all the divers systems, and with very little certainty in the diagnosis.

This scepticism, this incredulity, constitutes, in my opinion, anachronism. I say it persuaded of its truth; and it is this happy belief in the certainty of our actual means of exploration, and the disease, to proclaim aloud that which may place the science upon a footing that will no longer permit it to be said that it advances by hazard, that have induced me to add this small volume to those which have preceded it from others, in a great part from different and even contrary schools.

I am aware that among these last there are those who announce this confidence in the validity of their theories, and give the assurance that to them alone appertains the thread of Ariadne, without which the clinical study of the heart is a

labyrinth where one would be lost. Between them and us the public will determine. The dispute, it seems to me, is very well worthy of its interest.

I have been impressed by another idea in the composition of this work—a desire to simplify, as much as possible, the study which is so often truly arid and repulsive, thick set, as it is, with difficulties proper to it, and still more so with those that are added to it by a bad system of observation.

This small volume addresses itself, then, to physicians, who will find in it a collection of clinical facts, of which the analysis, verified by autopsies, will demonstrate to them to what a degree of precision the diagnosis of diseases of the heart is capable of attaining at the present time; and to students, who, in this *manual of diagnosis* will learn how we arrive at these practical results, the veritable conquests of contemporaneous clinic.



CLINICAL RESEARCHES  
FOR  
FACILITATING THE DIAGNOSIS  
OF  
ORGANIC DISEASES OF THE HEART.

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Of all those diseases of which the progress of medicine has latterly improved the diagnosis, but few can, in my opinion, compare with the organic affections of the centre of circulation. Such at least is the conclusion drawn from a series of observations that I have made in the wards of Professor Bouillaud, for near six consecutive years, either as a simple observer of the successful practice of this gentleman, or, during the last two years as *chef de clinique*.\* I would even say the diagnosis of these affections seems to me to offer a degree of certainty almost mathematical, if I did not fear that such an assertion, made so prematurely, would appear to my readers as an exaggeration.

And, indeed, the variety of opinions held by the majority of practitioners relative to the theory of the normal sounds of the heart, have this unfortunate result, that so far as several of them are concerned, the morbid sounds of this organ, to which especially, as we will hereafter see, diagnosis at present owes its certainty, are far from having that semeiological value which so properly belongs to them, and that some of them are at the present stage of science enveloped in mystery. One of those practitioners, whose talent for observation I am far from questioning, has said, "*that we can no more explain, by the theory of Mr. Roaunet, than by that of others.*"

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\* There is no English term, by which this can be translated. The *chef de clinique* is found only in those wards which are attended by a professor of clinical medicine; he is a graduate, and without residing in the hospital building has charge, under the professor, of the service.

*all the anormal sounds heard in the heart, and thus arrive at the diagnosis of the special seat of the disease."* (Chomel's *Pathological Générale*, 3rd. edition, 1841.

This assertion I shall submit to the test of facts. I shall endeavor to ascertain if, on the contrary, science does not possess means of exploration by the aid of which lesions of the heart may be diagnosticated, not only in their ensemble, but often decomposed by the observer, into their anatomical minutiae; and if the most faithful and most precise of those means, is not auscultation based upon the theory of Mr. Roaunet; a theory which, thus put in connexion with pathological facts, if it explains all of them, will receive a brilliant confirmation.

After this clinical part, I will commence a succinct study of the means of investigation, which may have furnished us the principal diagnostic signs; I will note their respective value, their semeiological bearings, in such a manner that those who in their turn may desire to investigate diseases of the heart, may find in this summary a kind of compendium of diagnosis. Finally, I will point out the practical utility of these investigations and show that far from being intended only to gratify foolish curiosity, they are susceptible of therapeutical applications of the highest importance. Moreover, I wish it to be understood that what I am about to publish are clinical researches. I will recur with the reader to a train of facts that we will examine together, gradually passing from the most simple, to the more complex, analyzing all with the same care, and receiving from some the where-with-all to clear up the history of others; and then, whilst all these facts will thus furnish us what they can of information and practical lights, in carefully collecting the results of this examination, in assembling these sparse materials, these successive acquisitions, we can not fail to verify the deficits which may exist. We will thus at the same time, alike discover what is possessed and what is yet wanting in the science; and this sort of inventory, if I may thus express myself, this comparative approximation of our wealth and of our poverty, will have, I hope, the advantage of showing some observers that we are less poor than they suppose, and of pointing out to others that which still remains to be acquired, so as forcibly to address the zeal of all, and direct more profitably the efforts of those who desire to devote themselves to the development of our art. Such is in short the essential design of this memoir.

## FIRST PART.

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### EXPOSITION OF FACTS.

Before commencing the clinical review, I think it necessary first to establish as succinctly as possible, the theory which shall direct us in appreciating the sounds of the heart. And here perhaps more than elsewhere, a minute knowledge of the normal state should necessarily precede the study of the anormal.

Four elements, in my opinion, should be admitted as entering into the production of the sounds of the heart.

1st. The shock of the apex of the organ against the parietes of the chest;

2nd. The gliding upon each other of the opposite surfaces of the pericardium;

3rd. The friction (*frottement*,) of the sanguineous column against the interior of the cavities of the heart;

4th. And lastly, the sudden readjustment of the valves, valvular clacking.

Of these four conditions, the last is the essential, the predominant. But, if the other three are habitually masked by it, if each of the three first is normally aphonic, it is nevertheless true, that, in pathological conditions, each can in its turn predominate. This is a fact susceptible of demonstration, and which it is especially necessary to bear in mind in the appreciation of the morbid sounds which occupy us.

Thus, 1st., *the shock of the point of the heart* habitually takes place, in my opinion, without any distinguishable sound; but in pathological circumstances we see this shock, by its exaggeration, produces sometimes a dead, dull, distinct sound, readily distinguishable, very different from the ordinary sounds of the heart, and sometimes a peculiar tinkling, (*the auriculo-metalick*,) a sound of but little symptomatic value, as we will hereafter see, and really compatible with the normal state.

2nd. *The gliding of the opposite surfaces of the pericardium.* So long as these surfaces are lubricated, humid, and perfectly polished, no sound results from their friction, but let

this polish become effaced, let false membranes be deposited upon these opposed surfaces, and there will immediately result a morbid sound which I shall here be content with having mentioned.

3rd. *The friction of the blood against the interior of the cavities of the heart.* As the last this friction is not in my opinion the cause, in the normal state, of a perceptible sound, the endocardium, a polished membrane, too readily facilitates this friction for it to produce marked sounds; but if this endocardium becomes partially thickened, or covered with vegetations, or encrusted with a chalky deposit, or its orifices become contracted, etc., all of these new conditions, by increasing the friction of the sanguineous wave, become so many powerful causes of anormal sounds.

It is to this cause that we must refer all the sounds produced by any derangement in our fourth condition, the *valvular clacking*, no lesion whatever of the valves producing any decided sound, save as a consequence of increased friction. One more word upon this clacking as it takes place normally, and we will have finished these preliminary considerations.

The action of the heart is divided into two successive and distinct times or sounds. Now what passes in the first time? Let us take, for instance, the left heart; whatever we may say of the left side being applicable to the right, whose movements are exactly synchronous. In the first time, the left ventricle contracts, and the greater part of the column of blood escapes by the open orifice of the aorta, whilst some little of it strikes the bicuspid valve, and thus adjusting it, stops all flow towards the auricle. It is this sudden adjustment of the bicuspid valve which produces the first sound.

Mr. Bouillaud adds to this re-adjustment, the collision of the valvular lamina, which he says are struck by those opposite. I acknowledge that it has always been difficult for me to appreciate this circumstance; doubtless because I explain with a slight difference, the mechanism of the play of the valves. According to Mr. Bouillaud, the adjustment of the auriculo-ventricular valves would be caused, not solely by the shock of the sanguineous wave, but principally by the action of the *chordæ tendineæ* which are inserted in the valves, and which would thus have a double use—1st, to replace and to stretch these moveable lamina, by exerting traction upon them from their circumference to their centre; and 2d, this re-adjustment having taken place, to prevent their inversion by the shock of the blood towards the corresponding auricle. Of

these two uses, I acknowledge that the second has always seemed to me to be the principal. For how are we to conceive that tendons fixed more or less perpendicularly to the ventricular face of the valves can be able, by contracting, to draw these valves in a horizontal direction? Is it not more natural to regard these *chordæ tendinæ* only as ingenious accessories, the necessary complement of a pump, both suction and forcing, but which is destined only to the support of one of the valves of this pump, to give it a solid prop, and a sufficient resistance against the action of the liquid which strikes against it? If this theory is true, the tendinous apparatus will be proportionably stronger, as the valve may have to sustain a more decided impulsion. This is what does happen; for we observe that the bicuspid valve is furnished after this manner, with the two most vigorous *columnæ*; and that it receives the insertions of about twenty-five tendons, the marked inferiority of the tendinous system of the bicuspid, being in proportion to the less powerful contractions of the right ventricle. Moreover, if a tendinous apparatus is necessary to valvular occlusion, why are the aortic and pulmonary valves unprovided for? Do not the arterial orifices require occluding as perfectly as do the auriculo-ventricular? Let us see in the *chordæ tendinæ*, only a means of resistance and not an arrangement for closing, and the absence that we have just noted will no longer astonish us, when we remember how slight the systolic force of the aorta and pulmonary arteries is, compared to the ventricular systole, how unnatural it is to displace from the direction of the ventricles those valves which the influence of the blood has just readjusted by its reflux movement after having displaced them by its issue. But this leads now to the consideration of the second sound.

Now what takes place in the second sound? The ventricle dilates, and thus gives rise to a tendency on the part of the blood to flow thither. But it can come from two directions: 1st, from towards the auricle, and there is nothing to oppose this, for the species of suction exerted by the dilating ventricle, depresses towards its interior the bicuspid valve, and the sanguineous contents of the auricle thus enter freely into the ventricular cavity; 2d, this suction, of which I have just spoken, is also exerted upon the blood already thrown into the aorta, a part of this blood then returns, thus obeying both the attraction of the ventricle, and the pressure exerted by the elastic walls of the vessel, but the return of this retrograde wave immediately gives rise to the adjustment of the aortic



valves, and this abrupt adjustment is the cause of the second sound. Thus then: the first sound, is the clacking of the bicuspid, the second sound, clacking of the aortic valves. Be it mentioned, however, more with reference to the pathological than to the normal heart, that at the moment when one of these valvular arrangements is adjusted, that when one of these orifices is closed, the other valvular apparatus becoming depressed, the corresponding orifice is traversed by a jet of blood, first, the aortic orifice, and in the second time, the bicuspid. And furthermore, let it be borne in mind, that this condition is itself so favorable to the production of a new sound, that even physiologically, if ever so little exaggerated, if, for instance, the column of blood should be expelled with a little too much quickness by the aortic orifice, an accessory sound can take place during the first time; as an example of this, we find sometimes, a slight, soft, aortic souffle, during the first time in persons who have just run or walked rapidly a short distance; a souffle that I have more than once found in patients at the moment of their arrival at the hospital, but which has disappeared after several hours rest. But it will be asked: if the swiftness of the flow of the blood, suffices for the production of a souffle, how comes it that this takes place only at the aortic, and never at the bicuspid orifice? Why should it happen only during the first time, and never during the second? I think that there are two reasons for this fact. The first is, that the blood should leave the heart more quickly than it enters, the ventricular systole which drives it out, being more powerful than the auricular systole which forces it in, aided as much as may be by the diastolic aspiration of which I have just spoken. The second reason is the difference in the normal dimensions of the two orifices, the circumference of the aortic being only 31 lines, and that of the bicuspid orifice  $46\frac{1}{2}$  lines, thus it is evident that the first is less able than the second to contract without a perceptible effect being the result.

In fine, let us remember that, of the two sounds of the heart, the first is naturally a little obtuse and prolonged, the second shorter and clearer; for in this difference there exists a new proof in support of the theory of valvular clacking. In fact, the first sound is obtuse, because the bicuspid and tricuspid valves have no ambient bodies to propagate their vibrations, except the fleshy walls of the heart itself; and because, moreover, the chordæ tendineæ, which connect with each of them, stiffened both by the contraction of the

columnæ carneæ, which put them in play, and by the fact of the readjustment of these valves, probably moderating, and in part extinguishing this vibration. The clearness of the second sound, results from conditions precisely opposite, in which we find the aortic and pulmonary valves, which are alike unprovided with chordæ tendineæ, and adhering to two arterial tubes, eminently elastic and vibratory. I come now to the clinical facts.

The number of organic affections of the heart of which I have collected observations, exceeds sixty. Desiring to consider here, these diseases principally in reference to diagnosis, I have felt obliged to put aside all those which have not received the verification of post mortem examination. This condition reduces the facts, that I am about to analyze, to about twenty.

In order to proceed, as I have already remarked, from the simple to the complex, I will begin with a case of hypertrophy of the heart, without a lesion of the valves, sufficiently prominent to have been remarked in the diagnosis. These cases are rare. We conceive, in fact, that the valves are parts so essential to the heart, that this organ cannot become hypertrophied to a certain degree, without these membranous vails participating with it; and after the details into which we have entered, it is readily perceived that in our opinion the least deformity, the least thickening of the valvular laminæ, should be expressed by sounds sufficient to be transformed into diagnostic signs.

**OBSERVATION 1.**—*Simple Hypertrophy without any remarkable valvular lesion. Œdema of the glottis.*—A laborer, aged 44 years, was received, on the 26th of September, 1840. He was of a medium constitution, and of a temperament more lymphatic than sanguine. He had labored for twenty-one years in a quarry, had been crushed by a fall, and his chest, at the time of his reception, was still a little prominent before, and notably depressed behind; seeming to carry thus the traces of an accident, which had detained him nearly four months in Saint Louis, where he was treated by the application of eighty leeches to the right side, and thirty to the neck. Five years had elapsed since he contracted what he called a catarrh; and without further specification in reference to this malady, he said it required the application of fifteen leeches to the præcordial region. Apart from these two epochs of his previous life, his health had been habitually quite good. Even at this moment he complains only of a slight

dyspnœa, which began eight days ago, a little cough without expectoration, and a cephalalgia with some dizziness; there is no other notable phenomenon, neither anorexia, fever, or even palpitations.

I examined him with care, and the following are the principal results of that exploration:

The digestive functions are well performed; temperature of the body normal; pulse from 40 to 44, of medium development, unequal, intermittent, irregular, the regular pulsation being sometimes followed by a small pulsation which immediately succeeds it; no præcordial prominence, nor anormal dullness in that region, nor vibratory tremor. The sounds of the heart are remarkable for the same anomaly of rhythm as the pulse, and still more so for their dry tone, somewhat like the rustling of parchment, but without souffle; they may be heard as far as under the right clavicle. The cough is quite rare, and without expectoration; no pain in the chest; the resonance and respiration are good before; behind idem, also on the right side, except at the base, where we hear a sub-crepitant râle with very numerous bubbles a râle; which is found in all the left side, accompanied on that side by a little obscurity in the resonance; a little cephalgia and heaviness of the head; a slight œdema of the inferior portion of the right leg; the patient was able to walk to the hospital without difficulty. With the symptoms which we have enumerated, before us, what shall be our diagnosis? An acute lesion of the respiratory apparatus? This idea is scarcely compatible with the number of the pulsations.

But for the present let us remark in anticipation, that in this case there was pulmonary œdema, a disease but little known, considered as an active malady, but little studied up to the present period, and of which, I confess, the idea had not occurred to my mind. Mr. Bouillaud who repeated the examination the next morning, remained in the same doubt on his part. Nevertheless, he prescribed a bleeding, to the extent of twelve ounces, a pectoral tisan, *un julep et le quart*. As to the heart, what had we a right to admit with the signs I have enumerated, except a medium hypertrophy without any grave lesions of the valves? And yet we could presume this hypertrophy only with a certain reserve, deprived as we were, and that perhaps by the accidental deformity of the chest, of its most ordinary signs: which are as we shall see, an increase of dullness, præcordial prominence, a displacement of the apex of the heart, and as we had here, in place of all



other symptoms of that morbid state, irregularities of the pulse and a parchment tone of the sounds, characters which we will find always responding to a certain thickening of the valves, and very rare without hypertrophy of the heart itself.

For some days the state of the patient remains stationary. The blood drawn is normal. A plaster of *pix burgund.* with an addition of *ant. tar.* was applied to the anterior portion of the chest. Yet, the stethoscopic phenomena persisted; the pulse became more regular, but it is also more tense, a little vibrating and has gradually risen to 60—64. At the same time the infiltration of the inferior extremities insensibly augments, and the scrotum has become œdematous.

October 16th. The patient complains of more pain. Since yesterday he has suffered with quite an intense affection of the throat; his voice is guttural; deglutition painful; the posterior part of the mouth very red, with swelling of the uvula, but no enlargement of the amygdalæ; countenance quite animated; heat of the skin moderate, but the pulse is at eighty with some intermittence. Prescribed thirty leeches to the neck.

At my evening visit, I found the patient seated upon his bed, laboring under a well marked dyspnœa, with the inspiration a little hissing. In ausculting the chest I heard only a rhonchus which appeared to emanate from a point in the larynx. There is considerable anxiety. The skin is hotter and the pulse from 96 to 100. The leech bites still bleed, nevertheless, I ordered a bleeding to the extent of sixteen ounces, and applied sinapisms to the feet. Notwithstanding these means the dyspnœa persisted, and the patient died at one o'clock, A.M.

*Autopsy.*—The epiglottis was remarkable for its apparently œdematous thickening, a thickening in which the adjacent parts participated, and especially the ary-tæno-epiglottidian ligaments. We incised these different parts, and there escaped from them a yellowish pus, recently formed, and infiltrating all the ambient cellular tissue. The glottis was sensibly contracted; the vocal cords were puffed, œdematous, without any trace of suppuration; the ventricular cavities were effaced; the laryngo-tracheal mucous membrane was very pale. The lungs, quite flexible and crepitant, were only engorged posteriorly and very abundantly infiltrated with serum. The heart was remarkable for its volume, the hypertrophy affecting especially the left ventricle. There was a slight thickening of the bicuspid valve. The aortic valves preserved their normal thickness; we remarked only that the tubercle of

Arantius of each of them, was rather more developed and harder than natural. The entire endocardium, the origin of the aorta and of the pulmonary artery were of a very lively red and faded only slightly by washing. This observation, which I have thought it my duty to reproduce entire on account of its interesting character, is calculated to give matter for numerous reflections, above all in relation to the œdema of the lungs; but do not let us forget that what we were particularly interested in was the diagnosis of the disease of the heart. Observe, then, that the autopsy fully verified the symptomatic indications. The signs noted during life were those of simple hypertrophy, they indicated scarcely any thickening of the valves. In this respect the necropsy sustained the diagnosis, neither exceeding or falling short of it. I will content myself with this remark, only adding in reference to the cause of the disease, that we may be permitted to hesitate between the external violence and the acute thoracic affection; but the second cause appears to me more probable than the first.

OBSERVATION 2.—*Left Auriculo-ventricular lesion exclusively, with Insufficiency.* Laurena Bavaux, laborer, aged thirty-nine years, entered into our service on the 22d of September, 1840. Her sickness dates six months past; she has tubercles in the summits of both lungs, and particularly the right side. After having satisfied myself in this respect, I passed to an examination of the heart. The patient says she is free from palpitations; the pulse is slightly developed, but this circumstance corresponds with the general emaciation; the præcordial dulness is normal; there is neither an increased prominence of the chest or vibratory tremor; I was then much astonished upon applying my ear over the region of the heart to hear a bruit de souffle sufficiently marked, seeming at first to be present alone in the second time, but becoming more and more pronounced, as I auscultated nearer and more near to the left auriculo-ventricular region, and in this point marking the two sounds and moreover not being continued either in the carotids or in the course of the aorta. With these signs I did not hesitate to announce the existence of a lesion of the left auriculo-ventricular valve exclusively, accompanied by a slight hypertrophy of the heart, the diseased valve should necessarily be patent. The double souffle I explain, by saying: the column of blood rubs against this thickened, puffed and imperfectly mobile valve, 1st. when a part of the column under the influence of the ventricular systole flows

back into the auricle through the imperfectly closed orifice (souffle of the first time;) 2nd. when the entire column, influenced by the auricular systole, and especially by the diastolic aspiration of the left ventricle, enters into this ventricle through the scarcely open orifice (souffle of the second time;) and if this second souffle is more marked than the first, it is from being caused by the friction of the entire wave of blood, the first being the result of the friction of a part of it only. From this difference of intensity between the two souffles, it resulted that the first and more feeble should become imperceptible at a short distance from its seat, and masked by the clacking of the tricuspid valve, whilst the second and stronger is heard even where the clacking of the aortic and pulmonary valves might replace it. Thus there is a blowing sound during the second time of the heart, throughout the whole præcordial region, and during both times in the region of the diseased orifice only. One month after her arrival at the hospital the patient died from the progress of the phthisis; and to speak only of that which now occupies us, we found at the autopsy, the heart of its natural size, perhaps a very little enlarged, when we remember the emaciation of the subject and the tubercular affection; the right cavities distended by newly formed clots; the tricuspid and pulmonary orifices perfectly healthy; the aortic valves likewise healthy, very thin and normal; the left auriculo-ventricular valve thickened, patent, it represents a kind of fibro-cartilaginous excrescence. Now, is not one such fact an irrefutable proof of the truth of our theory? Let us mention, in passing, this peculiarity, that certain grave diseases of the heart, sometimes exist without very marked functional disorder, and that in some degree, without the knowledge of those who are afflicted.

**OBSERVATION 3.—*Lesion of the Aortic Valves without Insufficiency.***—Ann Prudhomme, a washer-woman aged seventy, on the 19th of May, 1841, entered the wards of the charity hospital. She is a woman of strong constitution, sanguineous temperament, has been sick but three or four times in her life, and does not know of what diseases, but is certain they were not rheumatism or affections of the chest. For two months she has been subject to headache, restlessness, dyspnœa, palpitations, slight cough, and for the last three weeks, with swelling of the inferior extremities.

Upon her arrival I examined her carefully, and so far as concerns the circulation, found the pulse 60, moderately developed and regular. The præcordial region, covered in a

great part, by large mammæ, seems to offer neither increased prominence, anormal dullness, nor tremor. The beats of the heart are deep seated, slightly perceptible to the touch; the two sounds, difficult to hear on account of the fleshiness of the patient, are separate, but the first is accompanied by a souffle, rather rude, sufficiently prolonged, reaching its maximum in the region of the aortic orifice, and of the ascending aorta, and being propagated into the carotids without being there compounded with the *bruit de diable*. The patient complains of palpitations from time to time, even when in a state of repose; her sleep is often troubled by dreams, accompanied with starting. Her legs, and especially the right, are the seat of swelling, with tension and even a slight erysipelatous redness of the skin of the two inferior thirds—no ascites.

*What was our diagnosis?*

*A medium degree of hypertrophy of the heart, with lesion of the aortic orifice especially, without insufficiency of that orifice.*

Let us state, in few words, the grounds for the different elements of this diagnosis. And first, of the *medium hypertrophy of the heart*; there is *hypertrophy*, for, as we shall soon see, there is valvular lesion; and the one almost constantly supposes the existence of the other; *medium* for, there is neither vibration of the pulse, nor dullness, nor very sensible beats, etc.; *lesion of the aortic orifice*, for to what time does our souffle correspond? To the first time. Now what occurs during the first time? The left ventricle contracts, as we have said, and the column of blood escapes by the aortic orifice, without being accompanied by any other sound than that which results from the sudden re-adjustment of the bicuspid valve. We have here a souffle in the first time; what does it indicate? An excess of friction (*frottement*) of a part of the column of blood against one of the orifices of the heart; now, which is the orifice? Evidently, in the first time, there is as much probability of its being the bicuspid orifice incompletely closed, as the aortic orifice imperfectly opened. How then, shall we determine this difficulty? By examining with care the point with which the maximum of the souffle corresponds; particularly if this souffle extends towards the base of the heart, between the mammæ and the sternum, and is prolonged in the direction of the ascending aorta, as far as to the carotids, its point of departure is at the aortic orifice.

But we have said that the lesion was *especially* seated in the aortic orifice. Why then, this reserve, which seems to indicate the possibility of a lesion of the other orifice? It is



because rigorously, this complication is not entirely impossible. In ausculting beyond the bicuspid region, the embon-point of the patient did not permit us to analyze the sounds with sufficient precision, to be well assured that no trace of souffle mingled with the clacking of that valve. Hence we were justified in inquiring if a slight bicuspid insufficiency did not coincide with the aortic lesion.

Finally, we have said *without insufficiency of the aortic valves*. And, why? Because the second sound, that is to say, the result of the readjustment of these valves, was mingled with no souffle, which would have been inevitable, if a portion of the sanguineous wave had reentered the left ventricle, through the imperfectly closed aortic orifice.

This patient, who was very promptly restored to her habitual state of health, by repose and digitalis, prolonged her stay in our ward, where she shared the occupations of the infirmary. But at the end of about nine months, she was attacked with general acute bronchitis, and died on the 22d of February. During her long stay, I had carefully ausculted her, without even finding any modification in the seat of the souffle of the first time. The following is the result of the autopsy:

The heart is enveloped in a very thick bed of fat. After the removal of the fat, its volume is not much more considerable than natural; nevertheless, the auricles are dilated and the left ventricle evidently hypertrophied. The walls of this ventricle, towards its base, have a thickness of about thirteen lines. (The medium normal thickness is about seven lines.) Water poured into the aorta rises and preserves its level perfectly. Yet, the aortic valves, although sufficient and well formed, are incrustated with a hard osseous like matter, creaking under the scalpel, lineally disposed in a vertical direction to the valves. The internal face of the aorta is smooth and polished. The left auriculo-ventricular orifice appears normal; its valve, of a blackish red, from cadaveric imbibition, is well formed but a little thickened, especially in the part near the aortic valves. It is, however, neither fibrous nor ossified. The right valves are normal.

Is not this case exactly analogous to the preceeding, in the unity of the principal lesion, and in the perfect agreement of the diagnosis and the autopsy?

**OBSERVATION 4.—*Lesion of the Aortic Valves Exclusively, with Insufficiency.***—A woman thirty-six years old, a seamstress, was recieved into our service on the 11th of December,

1841, having an abdominal tumor, of a very obscure diagnosis, the history of which would draw us too far from our subject. Let us come then to that part of this observation, which, at this moment, must be of predominant and exclusive interest to us. I refer to the examination of the heart. Here again, as with the patient in our second observation, nothing seemed to point to the existence of an affection of that organ. There were no previous maladies, such as rheumatism, acute affections of the chest, palpatations or dyspnœa. Yet we heard with her, a very distinct double souffle, in all the præcordial region, but especially in the region of the aorta and its orifice; a souffle quite light in the first time, but more rude and prolonged in the second; and moreover, propagating itself into the carotids, with accompaniment of bruit de diable in the right. Further, the præcordial region, covered, it is true, by quite a voluminous breast, presented nothing very notable. The pulse is remarkable for its smallness.

The developements made in the preceding case, will enable us to dispense with any lengthy statement of the reasons for our diagnosis in this. Who does not perceive, 1st, from the seat of the souffle, that there is a lesion of the aortic valves; 2nd, from its existence in the second time, that this lesion is accompanied by insufficiency of these valves? Have we at the same time any considerable hypertrophy of the heart? No! doubtless, there is nothing which leads us to admit it. Let us, then, now form our diagnosis; *medium hypertrophy of the heart, thickning and deformity of the aortic valves, from which results their insufficiency*; let us add: *without notable deformity of the bicuspid valve*. Such are, in fact, the terms of the diagnosis, which I copy exactly from my observation.

Let us see now, what we will encounter in the autopsy, which was made nine days after the patient entered.

The heart is very voluminous, but it presents externally a tolerable quantity of fat, and in the interior some blackish, flabby, unorganized concretions. Freed from these clots and washed, it weighs 11 ounces, (about  $2\frac{1}{2}$  ounces more than its normal weight.) The left ventricle is nearly the exclusive seat of this hypertrophy: its walls are more than  $\frac{3}{4}$  inch thick towards the base. Nevertheless, the columnæ carneæ do not present a proportional development. Before opening this ventricle, water was poured into the aorta, and immediately it penetrated into the ventricular cavity. In fact the aortic valves are thickened, almost fibrous, especially at their free border, which is puffed, without adherences between them,

but a little shrunk upon themselves. The circumference of this is nearly  $2\frac{3}{4}$  inches. The internal face of the aorta, above the valves, presents some rugose, unequal plaques, of a very deep yellow—rudiments of cretaceous plates.

The bicuspid valve is well formed, scarcely any thickened, or rather, presenting only some small puffs, under the form of little reddish points. The circumference of the orifice is from  $3\frac{1}{2}$  to 4 inches. The auricle offers nothing remarkable. The same is true of the right side of the heart, which is perfectly normal. The circumference of the pulmonary orifice is 3 inches, and that of the tricuspid orifice is from 4 to  $4\frac{3}{8}$  inches. The perfect agreement which again exists here, between the diagnosis and the autopsy, has no need of being demonstrated. I will call attention only to the rugose plates which existed on the internal face of the aorta. These are causes of friction, (frottement,) and consequently of souffle, to be added to that, which the state of the aortic valves offers us. Let us take a passing notice of those small reddish points, disseminated on the borders of the bicuspid valves. I do not hesitate to consider them as so many fibrous concretions, plastic, fixed there, become adherent, living in some sort a parasitic life; and which, at a later period, would not have failed to become nuclei for cartilagenous and osseous formations, such as we are often presented with when valvular deformity has reached a high degree. In fine, let us remark the localisation of the disease in the left heart. To conclude, the orifices of this heart, (the left,) like those of the right side, have their circumference in exact proportion with the measures of the normal state established by Mr. Bouillaud.

**OBSERVATION 5.**—*Lesion of the Aortic Valves especially, with a cretaceous state of the Aorta.*—A porter, aged forty-eight years, of a strong constitution and sanguineous temperament, was recieved in our wards on the 27th of December, 1841. The only important peculiarity furnished by his previous history is, that in 1819, whilst serving in the cavalry, where he had been for eight years, he was kicked by a horse, on the anterior and outer region of the lower false ribs of the left side, and as a consequence was obliged to enter the hospital of Gros-Caillou. There treated by two or three applications of leeches, and as many of scarified cups; he came out at the end of two months, desiring to continue in the service, but was not able to stand the jarring necessarily consequent upon riding, being subject to palpitations and suffocation, on account of which he was discharged by Baron Larrey. Since

then, his health has been pretty good, except in 1821, when he was sick, but does not know of what disease—neither an affection of the chest nor rheumatism. Since that period, as before, his occupation has been laborious and fatiguing, and often caused him to experience, momentarily, violent palpitations and dizziness.

It is on account of these latter phenomena that he came to the hospital. Fifteen days before, there had supervened a decided infiltration of the superior and inferior extremities, of the genital organs, and even of the face.

*Condition upon admission.*—The face still presents some remains of the aforementioned puffiness. There is no ascites. The lower limbs are infiltrated. The pulse is 68, vibrating, developed, resisting—if I may so express myself, *hypertrophied*. There is a diffuse but evident præcordial prominence—a prominence established by the cyrtometre, (an instrument of which I will speak in the article on the means of exploration,) by a difference of two degrees. The point of the heart slightly elevates the sixth intercostal space, a little without the nipple. The beats of the heart are sufficiently expansive, but deep seated; and, at this time, without vibratory tremor. Dulness on percussion for three inches vertically, and three inches four lines transversely. Throughout all the præcordial region a double bruit de souffle has completely taken the place of the normal *tic-tac*. Towards the bicuspid orifice the two souffles are short, the second sharp, the first mingling with a dull sound, caused by the shock of the soft point of the heart. The nearer we approach the aortic orifice, the more pronounced do these two souffles become; that of the second time being more marked than that of the first; both are rude, uneven, very evident in all the track of the ascending aorta, as high as the clavicles, without valvular clacking in any part of the chest. The subclavian and carotid arteries present strong pulsations, with a propagation of the double blowing sound already indicated. There is also a simple souffle, only caused by pressure, in the abdominal aorta, and in the crural arteries. The jugular veins are well dilated. There is nothing remarkable in the respiratory apparatus, except a fine mucous râle at the inferior posterior part of the left lung.

Such were the signs collected upon his arrival, and confirmed the next day by Mr. Bouillaud, who added a vibratory tremor in the carotids and subclavian arteries.

*What was our diagnosis? Considerable hypertrophy of the heart, (one pound and upwards.) Thickening and defor-*



*mation of the left valves, with insufficiency of the aortic. Hypertrophy and chalky state of the aorta and arteries generally.* Let us now endeavor to specify with care the basis of this diagnosis.

*Considerable hypertrophy of the heart.* Vibrating pulse. Prominence. Displacement of the point of the heart. Diffused pulsations. Augmented dullness.

*Weight of the heart over one pound.* Its medium normal weight is from nine to ten ounces; we have here a præcordial dullness, at least twice as extensive as ordinary; we may therefore suppose the organ at least double its natural weight. Let us add, however, that these estimates never have a mathematical accuracy, especially when the weight of the heart surpasses, as we will see in this case, the ordinary limits of hypertrophy itself. In cases of this kind, in predicting what the heart will weigh, we mean that it will at least reach that weight and may possibly exceed it.

*Thickening and deformation of the left valves.*—As there is no valvular clacking, the valves are not only thickened, the lesion which they always first present to us, but deformed.

*Of the valves.*—Why both? Because we hear no clacking either in the first or second time, but in place of it, two souffles. Nevertheless, let us remark, that this double souffle is not always a proof of duplicate valvular lesion, as the same orifice, if it be insufficient, may produce a souffle at each time. It is evident, in fact, that in this case, the normal clacking of the other orifice may be masked by the souffle of the first, however slight this souffle may be. But if it is the nature of a souffle to be louder than the valvular clacking, it is much less capable of propagating itself to a distance. If, then, the clacking is smothered by the souffle, remove your ear from the point of departure of these two sounds, and auscult above the præcordial region, (and it will sometimes be necessary to do so, as high up even as the right clavicle,) and then the clacking will resume its place, and become perceptible, because the souffle will have ceased to be heard; and as soon as you can distinguish the precise times of this clacking, you may conclude, with assurance, the corresponding valve preserves its integrity.

There is, however, a case in which this precaution itself, this verification of præcordial auscultation, by means of conducting it a greater or less distance, may leave us in default. This occurs where there is a coincident valvular lesion of the right heart, and consequently a new souffle, with the

clacking which I suppose to be normal. This additional souffle, augmenting that of the left orifice, may propagate itself so far as no longer to permit, or only at rare intervals, the normal clacking to emerge into distinctness. This morbid coincidence, to which I now refer, will, in fact, present itself in the case now under consideration. We will see by the autopsy that there existed an insufficiency of the tricuspid valve; that thus it was a cause of souffle in the first time added to that furnished by the aortic orifice, and that these two souffles, by augmenting each other, sufficed to mask completely the clacking, supposed to be normal, of the tricuspid valve. I say *supposed* to be, since so far, there is nothing to warrant us in admitting the integrity of this valve. But I will suppress this restriction by adding that, during the residence of the patient at the hospital, the normal clacking became distinct by intervals at the first time.

I perceive this peculiarity noted for the first time on the 25th of February, in the following terms, dictated at the morning visit:

*Same sounds of the heart as on the first day. We finished the exploration by distinguishing the first sound on the lateral parts of the neck, and adjacent thereto.* We may perhaps be surprised to see a souffle, that is to say, the effect of a constant organic cause, thus vary in its intensity. But it is a clinical fact, daily observed, and of which I will give the reasons a little farther on, when treating of the stethoscopic signs.

I have stopped to develope, to some extent, the semeiological bearing of our double souffle, and the habitual absence of valvular clacking; and yet I cannot consent to quit the subject, without having responded to an objection, which might be addressed to me. A double souffle, I have said, does not indicate disease of two orifices, unless it is double at every point where it can be heard. If there be but one orifice involved, there will be a point more or less removed from the præcordial region where the normal clacking, marked nearer its seat, will reappear. Well! ought it not to be thus in all cases where the lesion of the left heart may be single or double? For, if the normal clacking cannot be furnished by either of the two left valves, may it not be by one at least, if not by both of the two right valves? How shall we distinguish this second case from the preceding?

I acknowledge that this difficulty may be met with. I believe that it is the cause of possible error in certain cases.

But I will add, that if this cause is not impossible, it is at least not frequent, as we will ascertain in the course of these clinical studies. This variety appears to me to be owing to the fact, that the normal sounds of the right heart are probably much less striking than those of the left; and this is; without doubt, a consequence of the habitual difference in the relative force of the contractions of the two ventricles, whether in a normal or morbid state. If this be true, it is apparent that these sounds may not be sufficiently distinct to be heard beyond the acoustic sphere of the souffles of the left heart.

Let us now return to our diagnosis. We have said, *insufficiency of the aortic valves*. Why so? For the twofold reason, that the maximum of the souffle corresponds with the second time, and to the region of the aortic orifice.

Finally, we have admitted with our patient *a chalky condition and hypertrophy of the aorta and of the arteries in general*. The vibration of the pulse, the force of the beatings of the sub-clavian and the carotids, the vibratory tremor of these last, cannot permit much doubt of the existence of a hypertrophy of these vessels. As to the cretaceous state of the aorta, we may be lead to suppose it, by the intensity and rudeness of the souffle in the aortic region, a souffle very intense, truly, to be the result only of valvular insufficiency, the degree of importance of which is somewhat restricted by the development of the radial pulse. Our patient after some alternations of momentary assuagement and suffocating paroxysm, was attacked, in the month of February, by an almost general œdema, complicated by erysipelas of the inferior extremities, and albuminuria, and finally died on the 2nd of March.

*Autopsy.*—The heart is enormously hypertrophied. Disembarrassed of its clots and washed, it weighs, with the origin of the large vessels, over one pound and a half.

*Right Cavities.*—The right heart participates in the hypertrophy, by which, however, the left is more particularly affected. Thus, the muscular substance of the right ventricle is firm enough and the columns quite robust. The dilated pulmonary artery, offers at its orifice a circumference of  $3\frac{3}{4}$  inches, (instead of about  $2\frac{3}{4}$ .) Its valves otherwise sound and well formed have a depth of nearly 10 lines, (instead of 6.) The auricle is sensibly dilated, and its orifice is 6 inches, (instead of  $4\frac{1}{2}$ .) in circumference. Moreover, its valve is sufficiently thin and well formed, but its size is not in proportion to the increased extent of the orifice which it should close;

for its maximum height is scarcely 1 inch, that is to say,  $2\frac{1}{2}$  lines more than usual; now the circumference of this orifice is about 2 inches too much, and the normal relation between the height of the valve and the orifice, being as 2 is to 10, it is evident that an excess, on the part of the orifice represented by 2 inches, would require on the part of the valve an increase, represented by 5 lines, and not  $2\frac{1}{2}$  as is here the case. The valve was then in default, that is to say, insufficient by a quantity represented by  $2\frac{1}{2}$  lines.

*Left Cavities.*—Water poured into the aorta seemed to maintain its level, or at best to penetrate but slowly, and the aortic valves thickened, hypertrophied, and even slightly fibrous, seemed not to have been insufficient. The circumference of the aortic orifice was near 4 inches and 4 lines, (instead of  $2\frac{3}{4}$  inches,) but the valves are proportionably enlarged, their height being near 10 lines, (instead of 6 or 7.) In the substance of the partition separating these from the pulmonary valves, was found a cretaceous production about twice as large as a cherry stone, difficult to enucleate, adherent to the aortic valve nearest to the pulmonary artery, leaving after its removal a little hole in the thickness of the aortic valve. Upon the inner face of the aorta, there was remarked some roughness, and even two small ulcerations, with irregular borders, from 1 to  $1\frac{1}{2}$  lines in diameter. In the thoracic aorta we found three small prominent yellow patches.

The left auricle was less dilated than the right. The bicuspid valve was healthy, possibly a little puffed upon its free border. A portion of this orifice having been cut, it was impossible to measure its circumference; but no matter how much it may have been developed, the valve was evidently sufficient, for its height varied from 10 lines to, in its maximum, even 15.

The left ventricle was essentially the seat of hypertrophy. The thickness of its walls, from the base to the point, is 1 inch.

The large arteries, and particularly the carotids and crurals, are hypertrophied; their walls, without being encrusted, are firmer and more resisting than normally. An almost perfect accordance between this autopsy and our diagnosis, is readily perceived; yet there are two points of disagreement, and especially one which I prefer to mention frankly myself. Thus the weight of the heart considerably exceeded our estimate. In reference to this matter, I will content myself by referring to the reflections which follow that estimation. A more essential point is the slight probability, or at least the



slight importance of the aortic valvular insufficiency, indicated by a souffle, rude, noisy, and much more evidently marked in the second time, than in the first. Here, I confess our error, remembering, at the same time, that we suspected the cretaceous state of the aorta, and I said that this souffle was not owing to aortic insufficiency. And I make this acknowledgment frankly, because it furnishes us, for the first time, a piece of useful information, which contributes to perfect what we have said above, on the souffle of the second time. It shows us that the souffles in the aortic region may be owing, not only, as is ordinarily the case, to the reflux of the blood into the ventricle, but also to the friction, (*frottement*) of the sanguineous column during its return shock, upon the inequalities with which the aorta is covered. This was the case with our patient. And who does not see that this friction is equally possible in the first time.

Further, this clinical point is so very important, that I cannot consent to pass it, without having demonstrated it by new facts, as precise as possible. We proceed then, to see the cretaceous state of the aorta expressed:

1st. By a souffle in the second time as we admitted in the preceding case.

2d. By a souffle in the first time.

3d. By a double souffle.

OBSERVATION 6.—*Souffle in the second time caused especially by a cretaceous state of the aorta.*—Fracois Laforge, sixty-five years old, laborer, was received on the 28th of February, 1842. This man complained, at his entrance, only of pretty severe epigastric pains, of three or four days duration, caused by having inadvertently swallowed a small quantity of aquafortis. I designedly omit the details relating to this accident, the consequences of which were fatal at the end of a month, to take up that which more especially interests us. Interrogated in reference to his previous history, our patient only said that he had suffered with an affection of the right side of the chest, fifteen years before. Since then, his health was good, but he continued to be subject to palpitations. Having reached, in our examination, the circulatory apparatus, we were first struck by the notable displacement of the apex of the heart. The finger perceived it with facility in the sixth intercostal space, a little without the *mammæ*, there was no increased præcordial prominence; no vibratory tremor, nor notable augmentation in the extent of the dulness. As to the sounds of the heart, a little unequal, a little less distinctly

struck, than naturally, they offered in the præcordial region, properly speaking, no perceptible souffle; but in the region of the ascending aorta, a very distinct souffle replaced the second time—a souffle very rude, and augmenting in intensity towards the superior part of the chest. Quite above the sternum, the second time itself, was accompanied by a little souffle. The pulse, not febrile, intermitted every five or six pulsations; it was but slightly developed, taking into account the very strong constitution of the subject, but it was hard and very vibrating.

With these signs before us, what ought to be our diagnosis?

In the first place we must admit that there is hypertrophy. The displacement of the apex of the heart, thrown without the nipple, and into the sixth intercostal space, is a sign in this respect, in some sort pathognomonic. But ought we to admit a considerable degree of hypertrophy? I think not in the absence of increased prominence, augmented dullness, etc. I say, then, there is a *medium hypertrophy of the heart*. Let us pursue this analysis. The sounds are unequal, less distinct than natural, but without souffle in the præcordial region; we hear them both there. At present we can say that the valves are injured, but their play is not prevented, and neither of them is sensibly insufficient. But, in the aortic region, we have two souffles, and one of them exists, especially, in the second time. This consideration causes us, in some measure, to lose sight of what we have established, in saying that *there was insufficiency of the aortic valves*. I know that we added *with a cretaceous state of the arch of the aorta*; I know that returning more than once to this diagnosis, with the students who then followed my evening visits, I insisted, before them, on the cretaceous state of the aorta, as a predominating lesion; showing them, in support of this localization, how very positively the maximum of the souffle corresponded with the aortic region; at the same time I could not forbid myself the idea that an insufficiency of the valves themselves, also contributed to this souffle. And yet I was obliged to ask myself: if aortic insufficiency exists here to a notable degree, why is the souffle not more distinct in the region of the aortic orifice itself? Why could I not perceive it in that point during the first time? Why is the first time accompanied by a little souffle, only at a point quite above the sternum? Would not all difficulties have been avoided, if I had admitted only the cretaceous state of the aorta, as the essential cause of the souffle in

question? But it may be said, if it be thus, if the *souffle* of the second time has not for its principal cause, the reflux of a certain quantity of blood into the ventricle, why then this feeble development of the pulse, which is not proportionate to the constitution of the subject, or the vibrations of the radial pulsation? This peculiarity might be explained by an aneurismal dilatation of the aortic arch, and this dilatation did, in fact, exist. It is apparent that our aortic insufficiency excludes, in our diagnosis, the necessity of this explanation.

Now let us enter into the details of the post mortem examination.

*Autopsy, the 31st of March, 1842.*—The heart is sensibly more voluminous than natural. Freed from its clots, and washed, it weighs, with the origin of the large vessels, over seventeen ounces.

*Right Heart.*—The right ventricle is not dilated; it seems even relatively contracted, the maximum thickness of its walls, towards the base, is scarcely  $4\frac{1}{2}$  lines. The auricle is a little enlarged; the auriculo-ventricular orifice has a circumference of  $5\frac{3}{8}$  inches. Its valve is slightly thickened. Nothing notable about the pulmonary artery. The circumference of its orifice is  $3\frac{3}{8}$  inches.

*Left heart.*—The left ventricle is the essential seat of hypertrophy. Towards the base its walls are nearly 1 inch thick, (nearly 5 lines, more than natural). The auricle is not sensibly developed. Its thickened valve, puffed in many points, presents a transverse extent of from  $3\frac{3}{4}$  to 4 inches, ( $9\frac{1}{2}$  to 10 centimetres.)

The aorta is remarkable for the volume of its arch, which is nearly double its normal size. Water being poured into this vessel slowly penetrates into the ventricle. In fine, the valves sensibly thickened, almost fibrous on their free border, are well shaped and free in their play. The circumference of the orifice, closed by them, is  $3\frac{1}{2}$  inches; that of the arch, at the commencement of its curvature, is  $5\frac{1}{8}$  inches. Between these two points, and in fact, through almost its whole length, the descending aorta is notably hypertrophied, firm, and thickened, its inner face incrustated with cretaceous plates or patches so confluent towards its origin, that no space can be distinguished between them, unless there be, in this same point, some small solutions of continuity, apparently veritable ulcerations of the internal membrane.

The heart and large vessels contain some sanguineous concretions, in part formed, anterior to death.

Let us, in a few words, review some points of this autopsy. In the first place I said *medium* hypertrophy; and I must frankly acknowledge that over one pound exceeds this estimate; but I will add, that it is very rare for a hypertrophy to reach such a degree of development, with so few signs of its existence. This is an exception that it would be well to note.

Now, after the preceding details, as to the inner face of the aorta and the state of the aortic valves, we cannot doubt but that the blowing sound was caused principally, not to say essentially, by the first of these causes. So, by a species of anticipation, justified by the autopsy, I have previously said, in referring the soufflé to this cause, that the rest would not offer any serious difficulty. We can readily understand how the seat of the soufflé did not correspond to the orifice of the aorta, but to the arch of this vessel; and how the pulsation in the radial was so feeble, the systolic effort of the hypertrophied left ventricle being in part neutralized by the aneurismal dilatation of the aorta. Still there are two things of sufficient importance to arrest our attention. How does it happen that the soufflé was stronger in the second than in the first time? How were the præcordial sounds exempt from soufflé, the valves of the left heart being altered? To the first question, I can only answer by an hypothesis, that is to say, by supposing that the aortic incrustations had such a direction or configuration, (that I did not notice it is true, but which is not at all impossible,) that the friction of the blood was greater as the wave returned, than when it jetted forth from the ventricle. To the second question my answer will be more formal, more solidly based upon clinical experience.

Every valvular lesion is not sufficient to produce a soufflé. As we have more than once said, this sound presupposes an excess of friction, for instance, a constriction of one of the orifices. But here we have seen that the orifices were sufficiently well formed, the valves were thickened, nearly fibrous, but free in their play, consequently they fell sufficiently to form no obstacle to the exit of the blood. But, it may be asked, will valves, thickened as these are, give a normal clacking? Let us recall that, from the first day, we noticed in the præcordial region sounds unequal, and less distinctly struck, than normally. I know, however, that thickening of the valves, when no soufflé is produced, does generally make itself known by a peculiar modification that we will discuss for the first time in the following case, I mean the parchment sound, (*bruit de parchemin*.) But this degree of sound seems to me



to require for its production a precise amount of thickening which it is impossible to determine exactly, but beyond which, the sounds gradually lose their distinctness, thus arriving by a kind of transition even to the complete effacement of all clacking, and the production of new sounds.

The case that I am now going to report, offers striking analogy to the two facts just mentioned. But independent of its diagnostic importance it affords another species of interest.

This case which I noted almost six years since, at the visit of Mr. Bouillaud will show us, that even at that epoch, the precision and aptitude of this able observer was not improvable. One can readily imagine that I should seize with avidity this occasion of paying a debt of gratitude to a master whose pupil I will ever feel proud of having been.

**OBSERVATION 7.**—*Souffle in the first time, especially with a cretaceous state of the ascending aorta, and the adjacent large arteries.*—George Thomas, locksmith, 48 years old, was received on the 13th of April, 1837, complaining, (especially for two months past, and without any important anterior malady, in an etiological point of view) of palpitations and panting, on the least exercise, of syncope or numbness, and of very intense headache.

*Actual state on the morning visit of the 14th.*—Evident prominence above and within the left nipple, prolonging itself slightly towards the superior part of the sternum. No vibratory tremor. Præcordial dulness over a space of more than  $4\frac{3}{4}$  inches in diameter. Impulse of the heart moderate. The apex beats 15 lines below the nipple. In the region of the heart, the two sounds of the valves are very dry, parchment like, especially the second; a light souffle accompanies the first towards the level of the aortic orifice. In rising towards the arch, this souffle becomes more intense, like filing, and tends to become sibilant, and is followed by a dry *parchment* clacking. The same souffle extends into the sub-clavicular region of the right side; we distinguish it also on the left, but more feebly. Towards the point of the heart, and a little below, the sound of souffle ceases. Above, and within the right clavicle, we perceive pulsation sensible to the eye, and a perfectly distinct vibratory tremor. There is a dilatation of the right jugular vein; a branch goes off to anastomose with the left which is not dilated. The pulse of the two radials is sensibly the same, although perhaps a little more developed in the right; it is at sixty-four, regular, wiry, gradua-

ted and vibratory. The coats of the crural arteries, are like cartilage with some inequalities on their surfaces; their pulsations are vigorous and resisting. Under a rather strong pressure, their sound is transformed into one of very pronounced souffle; under a moderate pressure there is a jerking sensation. We feel there no vibratory tremor.

**DIAGNOSIS.**—*Hypertrophy of the left heart, especially of the ventricle, with dilatation. Thickening of the valves. Dilatation of the aorta, of the brachio-cephalic trunk, of the origins of the sub-clavian and of that of the right carotid, with a cretaceous state of the arteries. General hypertrophy of the arterial system, with cartilaginous induration of the wall, particularly those of the crural.*

The same evening of his entrance, this patient, with no indication of so speedy a dissolution, complained of chills along the back, and regained his bed with considerable suffocation; and notwithstanding the aid of bleeding, died at half past 10 o'clock in the evening.

**Autopsy.**—Surface of the heart not covered by the lungs, is  $4\frac{1}{2}$  inches transversely, and  $4\frac{1}{4}$  vertically. The left ventricle, considerably hypertrophied, constitutes about seven eighths of the whole organ; its cavity is dilated, and its walls are nearly 15 lines thick at the base, and nearly 5 lines towards the point. The bicuspid valve is well formed, but thickened; the tricuspid is opaque, a little thicker than natural, and is roughened by some small tubercle like productions. The valves of the pulmonary artery are thin and normal. The aortic valves are thickened, presenting also some tubercular productions, but they are sufficient and not deformed. The aorta, covered by the lung in all its extent, is considerably dilated. Its coats are at least triple their normal thickness, and this thickening is continued along the descending aorta. On the internal face of this artery, especially towards its origin, are numerous calcareous plates; and further, in the region of the arch are some redness and rents, which we also find in the arteria innominata. This artery is also dilated, but in a less degree, as are also the sub-clavian and carotid of the right side. The thickening and cretaceous state of the arteria innominata ceases at the origin of the sub-clavian and carotid. The right radial artery is a little more developed than the left. The crurals do not present any plates on the interior, but their coats are thickened.

I ask, when we compare these necroscopic results with the details of the diagnosis, if one is not tempted to suppose that

the latter were specified after a sight of the former. I will ever remember the admiration of the assistants, and I may also say, my own astonishment at the sight of this sort of divination, to which a long experience had not then given the key, and which appeared to all of us so much more marvellous, as the diagnosis, so detailed, so precise, and so promptly proved by the necropsy, was in some fashion the impromptu of a single exploration.

I will make on this autopsy, only a very few reflections. It was in this observation that we encountered, for the first time, the dry *parchment* sound; we perceive the thickening of the valves corresponding with these sounds. A light soufflé, towards the aortic orifice, accompanies the first sound. We have seen that the aortic valves were not only thickened, but also roughened by small vegetations, fibrinous rudiments, deposited there, without doubt, by the coagulation of the blood, more rare, as we have already said, on these valves, than on those to which are attached the chordæ tendineæ. But as the tricuspid valve also presents these vegetations, I may be asked why it does not also give a soufflé, if not in the first time, since it was sufficient, at least, in the second? Because this soufflé, if it did exist, must have been so light as to be masked by the clacking of the aortic and pulmonary valves, and moreover deeper seated, in as much as it is a soufflé of the right heart, than were it one of the aortic orifice.

The farther we got from this orifice, in following the arch of the aorta, the more this blowing of the first sound predominated. Then it was in the artery, and not at its orifice, that its cause resided. But why should it not exist during the second time? Here I must make the same answer as in the preceding observation.

Here, in fine, we again find the pulse vibrating and but little developed, wiry; vibrating, because of the hypertrophy of the left heart; wiry, because of the aneurismal dilatation of the aorta. It then stands demonstrated, by this observation, that the aortic soufflé of the first time may be caused by the condition of the vessel itself, rather than by the state of the valves. I have proved by the preceding observation, that the soufflé of the second time can have the same cause, and consequently simulate patency of the aortic orifice. Now let us see if this same chalky state of the aorta, cannot give rise to a double bruit de soufflé. The following case will teach us.

**OBSERVATION 8.**—*Double soufflé from aneurism, and creta-*

*ceous state of the thoracic aorta.*—I have published in the *Revue Médicale* (May number, 1841) the detailed observation of an aneurism of the thoracic aorta, which offered this particularity, a development from before backwards, giving rise to a prominence, under the form of a double tumour in the region of the back. The entire absence of prominence, except a slight arching of the præcordial region, the increase of dulness of the heart, the obscurity of its two sounds, caused us at first to diagnosticate upon the admission of the patient, March 20th, 1838, an hypertrophy of the heart, with slight thickening of the valves without patency. A slight friction sound, believed to be pericordial, and which specially existed in the second time, caused us moreover, to suspect the existence of some plates or adhesions of the pericardium.

In a short time pain supervened in the region of the left scapula, and the examination of this part showed a pulsation that raised the fingers with force, and produced upon auscultation, a sound of collision, accompanied by a slight souffle. From that time the diagnosis could be completed, and we added: *aneurismal dilatation below the arch of the aorta, with patches (plaques) in the artery.* The tumour, the first indication of which we have just signalized, at first developed itself slowly. At the end of the first three months, it was even pressed down, as it were upon itself. At this time we heard in the heart a double bruit de souffle, dry, rasping, with its maximum in the first time, and towards the aortic orifice.

Thus it continued during the long duration of this painful affection which terminated in death on the 18th September, 1840. For my part I have often verified the existence of this double souffle, as often at the posterior part of the trunk, as in the præcordial region itself, even until, as a consequence doubtless of the thickness of the fibrous layers amassed in the interior of the aneurismal pouch, this double bruit became so obtuse, so distant, that it ceased almost entirely to be heard.

I will not here repeat the circumstantial details of the autopsy; but I will mention what is at this time for us the most essential feature of the case, that the aorta alone gave us the key to the double bruit de souffle, for it was dilated in such a remarkable way, that I caused the specimen to be moulded, and it was moreover rough, thickened, and riddled with fibro-cartilaginous, and even osseous plates. As to the heart, besides its hypertrophy, it offered no other valvular lesion than a slight thickening of the bicuspid valve, and especially of the aortic, but without deformation of these valves, or any considerable relative dilation of the corresponding orifices.



We perceive by the preceding facts, that certain lesions of the aorta, and particularly a cretaceous state of its arch, may simulate, in the stethoscopic signs, some valvular lesions. It is a cause of error, of which it is well to be warned, and against which we have seen that it is not impossible to guard. A question to which we are naturally brought by this difficulty is, how shall we diagnose the cases in which valvular and aortic lesions coincide? Is this double diagnosis possible? Yes, doubtless, I avow it in advance, and besides, we have already seen it in our fifth observation. But in those cases only, when each of these lesions presents a certain degree of importance, unless there be a marked predominance of aortic lesion, the advantage in a semeiological point of view, will always be on the side of valvular lesion. This we will now proceed to demonstrate by facts.

**OBSERVATION 9.**—*Concidence of valvular lesions, and a cretaceous state of the aorta.*—Lebbois Jean-Baptiste, printer, aged 47 years, entered into our service on the 14th September, 1841. The subject, for twenty years past, of rheumatic pains, generally apyretic. He had intense catarrhs, often accompanied by fever and paroxysms of dyspnoea, with palpitations, which at first separated by long intervals, gradually became more frequent, and for six months past, were reproduced by the least exercise.

*Present condition.*—General pallor, The face is neither puffed or discolored. Slight œdema of the inferior extremities. Cutaneous heat normal. Pulse 112, developed, vibrating, strikes forcibly. No very notable præcordial promiency. The apex of the heart perceptibly elevates the sixth intercostal space, but within the nipple. The impulsion of the organ, is moreover extended, without vibratory tremor, and the dulness is nearly 3½ inches square. In ausculting the præcordial region, we note at first, three very distinct phenomena: a heavy and very dull sound in the first time, the result of the shock of the point of the heart; effacement of the two valvular clackings, and especially of the second; and, in fine, a souffle. In the region of the left auriculo-ventricular orifice, this souffle is little pronounced; it is distinguished especially in the first time, whilst in the region of the aortic orifice, and particularly of the ascending aorta, it exists during both times, particularly in the second. It propagates itself but feebly into the carotids. The pulsation of these arteries is very strong, are perceived in the abdominal aorta, and especially in the crurals, of which the sound gives to the ear

the sensation of a very detached fillip. No distension of the jugular veins. Nothing very notable in the respiratory apparatus, except a subcrepitant râle, very fine and numerous, behind, and at the base of both lungs.

The next morning M. Bouillaud, who had requested me to make the visit, verified what we have just indicated, without adding any thing, and left me in charge of the diagnosis. I then dictated it in the following terms:

*General and very considerable hypertrophy of the heart, (about one pound.) Thickening and induration of the left valves, and especially of the aortic, without a contraction of the corresponding orifice, or insufficiency of the bicuspid valve.*

Why do I not add to this diagnosis, *insufficiency of the aortic orifice?* Because it would be contradictory to admit this insufficiency with a pulse so developed, and so forcibly struck. But from the moment that I did not attribute the souffle of the second time to this cause, was I not compelled, in order to explain it, to admit the existence of a cretaceous state of the aorta? Yes, without doubt, I confess the omission. But I go further, should I not have admitted, at the same time, both these lesions? For it appears to me, from observation, that a cretaceous state of the aorta implies, generally, a certain degree of hypertrophy of the coats of the aorta itself. Would not this hypertrophy explain those qualities of the pulse, which seemed to me incompatible with a reflux of the blood into the ventricle? Yes, if the pulse had been only vibrating—no, if the vibration was accompanied by a very considerable development; at any rate, I could reconcile these two elements of diagnosis, only by admitting an aortic insufficiency, little pronounced. It is evident that it is this same development of the pulse which leads me to exclude an aortic contraction, and bicuspid insufficiency. If this hypertrophy of the left ventricle could always explain the vigorous pulsations of the radial and carotids, there would still remain those of the abdominal aorta and the crurals, which, as we have already seen, and as we will see again, do themselves imply a certain hypertrophy of the arterial system, that I ought also to have specified. This is another omission which I will not seek to justify.

Let us return to our patient. Notwithstanding the use of digitalis, diuretics, præcordial vesication, ect., he grew gradually weaker. The infiltration increased; it affected the scrotum and penis to a degree that impeded the excretion of the urine. The pulse remained quite vibratory, but it lost its

volume. I found, at intervals, a slight vibratory tremor in the præcordial region; as to the souffle always replacing perfectly the second time, it became sensibly more contracted, constantly attaining its maximum in the region of the aortic orifice, much less distinct in that of the bicuspid orifice, and manifestly propagating itself into the carotids. The first time was quite dull and imperfectly struck, and perhaps even accompanied, towards the bicuspid region, with a little souffle. He died on the 17th of November.

This patient having been to me the object of clinical study, frequently repeated, at my afternoon visit, on the eve of the autopsy, I made before my pupils, a resumé of the diagnostic signs which we had noticed together in our last explorations, and I rectified or completed in writing, before them, my first diagnosis, thus:

*General and very considerable hypertrophy of the heart, (at least about one pound;) the hypertrophy affecting, especially, the left ventricle, of which the walls must be very much thickened; medium dilatation of the auricles, and of the auriculo-ventricular orifices. Thickening and induration of the left valves. A little restraint in the play of the bicuspid valve, without any very notable insufficiency. More considerable deformation of the aortic valves, which imperfectly close their orifice, and perhaps present some small vegetations. No considerable effusion in the pericardium. Perhaps some pericardiac plates. Pulmonary œdema.*

Let us show briefly, the reasons for this definite diagnosis.

*Hypertrophy, etc.* Displacement of the point of the heart. Expanse of impulsion and increased dullness.

*The hypertrophy affecting especially the left ventricle, etc.* The pulse in general, vibrating and forcibly struck. *Bruit de choc* of the point of the heart, very heavy and dull.

*Medium dilatation of the auricles.*—*Dilatation*, because 1st, generally in all cases of any considerably hypertrophy, there is a dilatation of the auricles; 2d, infiltration of the extremities, of the penis, and of the lungs, producing a stasis of the blood, which gives rise to dilatation of the auricles; these losing in contractility, what they gain in capacity; or, there may be a stasis of the blood from the contraction of the aortic orifice, or in consequence of bicuspid insufficiency; but here we would have neither; therefore, I might have added that it was especially the right auricle which was dilated. *Medium dilatation.* Because in great dilatations, the pulse is very small, and it is not so here. *And of the auriculo-ventricular orifices.*

When the auricles are dilated, the orifices ordinarily participate proportionably.

*Thickening and induration of the left valves.*—Effacement of the two valvular clackings.

*A little restraint in the play of the bicuspid valve.*—Effacement of the first sound, with a little souffle, perhaps bicuspid.

*Without any notable insufficiency.*—This souffle is very slight, I might even probably have attributed it to the tricuspid orifice; moreover the pulse is developed and vibrating.

*More considerable déformation of the aortic valves.*—The souffle attains its maximum in the aortic region, and propagates itself into the carotids.

*Which imperfectly close their orifice.*—Because of the souffle in the second time, by the return of the blood from the aorta into the left ventricle. I did not have, this time, so many reasons for admitting a cretaceous state of the aorta, as in my first diagnosis, but if I had made this diagnosis at first, I would have persisted in it.

*And presenting, perhaps, some small vegetations.*—Because there has been at intervals, a little vibratory tremor. If I had admitted cretaceous plates, I would not have made this supposition.

*No considerable effusion in the pericardium.*—The sounds are not distant from the ear, and there is no considerable prominence.

*Perhaps some pericardial plates.*—In the præcordial region, towards the base of the heart, on applying the ear, I often heard a sort of dry râle, of which I did not, at first, dare to affirm the nature and seat, supposing that it might be a bronchial râle; nevertheless, its persistence, difficult to seize, on account of the sounds of the heart, but which appeared to me independent of the respiratory movements, ended by giving grounds to suspect a pericardial friction, (frottément.)

Finally, *Pulmonary œdema.* Sub-crepitant râle towards the base of the lungs.

Let us now proceed to the autopsy.

The pericardium contains a very small quantity of serosity. On the external face of the heart, towards the middle of the height of the right ventricle, we remark two or three pericardial plates, forming a relief little pronounced.

The heart is considerably distended by recent clots, which especially fill the right cavities, and give to the whole organ a globular aspect. Its apex is soft and rounded. Well washed, it weighs with the origin of the large vessels, over



one pound. The hypertrophy occupies, particularly, the left ventricle, of which the walls have a thickness of about 10 lines.

The left auricle is but little or not at all dilated. Its bicuspid orifice has a circumference of  $9\frac{1}{2}$  lines.

The right auricle, on the contrary, is much dilated. It might contain a medium sized orange, The circumference of the tricuspid orifice is five inches, (instead of  $4\frac{1}{2}$ .)

The bicuspid valve is a little thickened, but quite sufficient.

The tricuspid has its normal thickness.

The enlarged aortic orifice presents a circumference of  $3\frac{1}{2}$  inches, instead of  $2\frac{1}{4}$ .

The sigmoid valves are very thick, fibrous and insufficient; for water poured into the aorta, penetrates the ventricle. The aortic orifice is, moreover, bordered on the aortic side, and to the height of about 14 lines, by cretaceous plates, quite prominent, creaking under the nail, which raises them with difficulty, representing a sort of unequal, wavy ribbon; we also find some of these plates in the descending aorta.

The pulmonary artery is sound, the circumference of its orifice is nearly  $3\frac{1}{4}$  inches, its valves are thin and well formed.

The right ventricle is a little dilated, and its walls are less than five lines in thickness.

The lungs present a general and decided serous engorgement, an abundant spumous serosity issuing from the surfaces of the incisions upon the slightest pressure.

I omitted assuring myself of the hypertrophy of the arterial system.

Notwithstanding this hiatus, which I regret, and the before mentioned omissions in my diagnosis, I ask every honest observer, if there are many organs in the economy, whose morbid alterations we can analyze in advance, and decompose their diseases into their different elements so minutely and with so much precision? But still some discrepancies existed between our last diagnosis and the necropsy. Thus I was wrong in saying dilatation of the *auricles*, the right auricle alone being dilated. I explained the effacement of the first sound, and the slight souffle heard during the first time in the left auriculo-ventricular region, by a constraint in the play of the bicuspid valve, the autopsy showed us that this valve was free, only a little thickened; so I prefer thinking that this moderate souffle came either from a little insufficiency of the tricuspid orifice, or as is more probably the case,

from the aortic orifice, in the region of which it was so evident.

The cretaceous state of the aorta, the study of which has been the peculiar object of the last four observations, has thus led us into a kind of digression, which I am far from believing useless, but which we cannot longer pursue without losing sight of the methodical order which we wish, as much as possible, to follow in these clinical researches. So now let us recur to the properly so called lesions of the heart. We have passed in review, the alterations of its two principal orifices separately; let us now take up those cases in which the two orifices are simultaneously affected. During the study of these, out-of-the-way and unexpected clinical deductions may possibly present themselves; we will not refuse to answer to these calls, but will only do so when these sorts of digressions are authorized by the importance of the object.

**OBSERVATION 10.**—*Double contraction without patency of the aortic orifice.*—Sophia Boulleau, a seamstress aged 17 years, was received on the 25th of March, 1841. She dated the commencement of her disease ten months back. About that time she was seized with an acute general articular rheumatism, which confined her to her bed for nearly two months, and since then she has suffered from palpitations of the heart, which she says had their commencement during this disease. In the course of last February there was an addition of pain, with swelling of the abdomen, slight diarrhœa and cough. These last symptoms decided her to come to the hospital.

*Present condition.*—Pallor, anemia, and general emaciation. Considerable ascites. Heat of the skin rather greater than normal. Pulse 124, very small and readily compressed. Palpitations even in a state of repose. A little præcordial prominence expressed on the cyrtometre by a difference of about three degrees. The pulsations are strong, visible to the eye, and extend, as does the dulness, from the fifth intercostal space, almost immediately below the breast, up to the second intercostal space above, the hand there perceiving a third concussion due to the abrupt passage of the column of blood without, strictly speaking, cats purr, (*fremissement cataire*.) The ear applied below the breast, distinguishes a double bruit de souffle, rude and rasping, without valvular clacking; above the breast, and especially in approaching the left clavicle, we can only hear a souffle in the first time, and the second sound is then made up by a hard,

quick, and essentially parchment like clacking. The souffle of the first time is continued into the carotids unmixed, at this time, with the (bruit de diable) sound of the humming top. The beats of the left sub-clavian artery are visible to the eye. The jugular veins are sensibly developed. The respiration is rapid, difficult, without any very notable morbid bruit, except a slight moist râle, very fine, at the base of the left lung. The feet are œdematous.

The ensuing morning, the 26th, Mr. Bouillaud, verified in his turn, the preceding signs, and he dictated the following diagnosis.

*General hypertrophy of the heart, (12 to 14 ounces.) Thickening and probably vegetations with deformation of the left valves.*—Let us analyze this diagnosis and see if our antecedent observations will not permit us to add to it still some accessories.

The hypertrophy of the heart, the exaggeration of its weight, are too evident for it to be necessary that we should now repeat the reasons.

*Thickening and probably vegetations of the valves of the left side.*—Why of the valves? I say that this double lesion is evident. And in fact, we have in the region of the aorta, a rude blowing sound in the first time, and this sound extends into the carotids, then the aortic orifice is diseased; but in the præcordial region, properly so called, we have two souffles; whence comes the second? Is it from this same aortic orifice, patent and consequently incapable of producing a valvular clacking? No; for in approaching the clavicle we reach a point where is heard a second clacking, harsh, quick, like parchment, evidently produced by the thickened but sufficient valves of the aorta. Then the bicuspid orifice is the seat of this second souffle.

We have said that the hand perceived a rather rude concussion, which appeared to be owing to the abrupt passage of the sanguineous column? What does this concussion indicate? A prominent obstacle to the escape of the sanguineous wave through the aortic orifice. What may constitute this obstacle? Is it a simple contraction? Then we should have, in the first time, a peculiar souffle, contracted and fine. But the souffle does not present this character; it is rude and unpolished. Is it a cretaceous state of the sigmoid valves? This is possible; but generally this cretaceous state is complicated with that of the aorta; but here we probably have no plates in the aorta, since there is no souffle in the region of this

artery, that which commonly occurs. It is more likely, then, that we have some vegetations about this orifice, and perhaps, (but I give this as a mere gratuitous supposition,) about the opposite orifice.

Let us now remark that the analysis of this fact alone brings us to add to the diagnosis a new element, viz: sufficiency of the aortic valves.

What shall we say of the other orifice? Nothing positive. Nevertheless, it is probably not greatly insufficient. For, how should we then have, by the escape of the sanguineous column through the aortic orifice, that rude concussion, which moreover announces that the hypertrophy is seated especially in the left ventricle? But will it be said that the pulse is very small? With a young girl, so emaciated, this circumstance is of little importance; yet it may be a reason for supposing a little contraction of the bicuspid orifice or of the aortic itself.

But shall we say nothing here of the right heart? Yes, without doubt, with a hypertrophy so considerable; with so many signs of a notable restraint in the venous circulation, the ascites, the distension of the jugulars, the serous congestion of the lungs, it is very probable that the right auricle is distended, if only passively, and that, on that side, we will encounter a dilatation of the auricle, and perhaps of the corresponding orifice.

We were promptly enabled to verify our diagnosis. The infiltration progressed, then acquired an enormous volume, to such a degree, that the heart was pressed upward, until we perceived it beating in the first intercostal space, and notwithstanding two paracenteses which produced only momentary assuagement, the patient succumbed on the 18th of May.

*Autopsy.*—I proceed to that which concerns the heart. Freed from its clots, this organ weighs over  $1\frac{1}{4}$  pounds. The hypertrophy affects the right and left cavities. The right ventricle is fully a third larger than natural; the thickness of its walls is about 5 lines in its medium part.

The tricuspid orifice is large in proportion to the ventricular cavity. The laminæ of its valve are thickened and perhaps a little too short to close the dilated orifice. There are some small vegetations on the free border of this valve.

The auricle is dilated in the same proportion. Its columnæ carneæ are very strong. There is nothing very notable about the pulmonary artery.

Water poured into the aorta reflows immediately; and in



fact the three valves are well formed, of at least triple their normal thickness ; but on the ventricular side of their free border, there is a train of small warts or cauliflower vegetation. The aortic orifice is small, absolutely and relatively, to the magnitude of the ventricle.

This is vast ; it might contain an ordinary sized egg ; its walls are 15 lines thick towards the base. The two columns of the bicuspid valve are enormous, one of them is as large as the ring finger.

This valve is very thick and hypertrophied. The orifice is quite well formed, yet, the angles of the valve are united by the agglutination of their opposite edges. All the free border of this valve is studded by many vegetations, gravelous and resembling the cauliflower. The cavity of the left auricle is dilated in proportion to the ventricle. Its walls are also thickened.

The mitral and aortic orifices are both contracted relatively to the augmented capacity of both the auricle and the ventricle.

I do not think it necessary to make any lengthy comments on this autopsy. I can but regret that the dilations were not expressed numerically. But I will say, in order to shelter from all suspicion my reputation as editor, that this autopsy, such as I have detailed it, was dictated in the amphitheatre by M. Bouillaud himself.

**OBSERVATION 11.**—*Deformation of the valves without Contraction.*—Joseph Doumère, shoemaker, aged 48 years, entered on the 28th of October, 1841. This patient, heretofore treated in this service, reports that although habitually healthy he was attacked nine years ago with febrile articular rheumatism, which affected all the principal articulations, and of which he was cured at the end of three months.

About twenty months since he began to experience palpitations, which gradually increased and forced him for the last nine months to discontinue his occupation. Since that epoch he has not been able to remain in bed during the night, and moreover, very frequently passing entire days in an arm-chair. During the latter months a notable infiltration, of the inferior extremities and abdomen, occurred. He had, also, during four or five months past, some symptoms of sero-sanguineous congestion of the lungs, such as a stitch in the side and very rusty expectoration, symptoms which were combatted by two bleedings, two applications of leeches, and numerous vesications.



*Present condition.*—Almost seated in bed, the patient is a prey to a very intense dyspnœa, his respiration being sensibly accelerated and his responses very brief. His face is pale, a little puffed; the lips slightly bluish; the abdomen is remarkable for its volume, and is a little sensible in consequence of the distension of its walls. The least shock produces the most evident fluctuations, and it is only at the distance of about one hand's breadth above the umbilicus, which is almost entirely effaced, that percussion ceases to produce a dull sound. The semi-circumference of the abdomen, passing the umbilicus, is nearly two feet. The urine is passed with much difficulty, the penis being infiltrated in all its length and twisted upon itself. The secretion is also remarkable for the infiltration of its coats. The limbs, even the superior, participate in this condition, the thighs and legs are of a considerable volume.

The pulse, difficult to feel on account of the œdema of the wrists and its smallness, is evidently disproportioned to the force of the subject, sufficiently equal, but intermittent and from 116 to 120. There are six degrees of precordial promineney by the cystometre. The point of the heart beats in the fifth intercostal space, but a little without the nipple. From this point, the pulsations extend as far as two or two and a quarter inches above the nipple, the heart being, moreover, perhaps pressed back by the abdominal effusion; they (the pulsations) are visible but in moderate force. The dullness corresponding to the region of the heart is from  $2\frac{3}{4}$  to  $3\frac{1}{4}$  inches vertically and from  $3\frac{1}{4}$  to 4 transversely. There is no vibratory tremor. The sounds of the heart a little masked by the interposition of a lamina of the lungs, are besides imperfectly struck, without distinct clacking, and at the same time without notable soufflé at this moment, nevertheless in proceeding along the ascending aorta, we distinguish in the first times, a sort of creak, very distant, and short, and which may be followed into the precordial region as far as the aortic orifice itself, but does not propagate itself in the carotids. These last arteries pulsate only very feebly. The jugulars are notably distended. In the respiratory apparatus we note a little diminution of sonority toward the base of both lungs, and a desseminated subcrepitant rale quite numerous, especially in the left.

M. Bouillaud being then absent, I dictated the following diagnosis.

*Considerable hypertrophy of the heart (about  $1\frac{1}{2}$  pounds).  
Relative and absolute dilation of the auricles. Thickening,*

*deformation, and perhaps adherences of the left valves, especially without very notable absolute contraction or vegetations.—Perhaps diminution of the pulmonary orifice in particular.—Slight effusion in the pericardium. Œdema of both lungs. Ascites and anasarca.*

Twelve days after, we were able to submit this diagnosis to the verification of an autopsy, the punctum which had given issue to a great quantity of fluid, having procured only a momentary assuagement. As to the sounds of the heart, they remained the same, save a little purring of the first time, which was at first only reproduced at intervals and finally entirely disappeared.

*Autopsy*, on the 4th of November.

*Thoracic cavity.* Œdema of both lungs behind and below. The heart is uncovered to the extent of about  $5\frac{1}{2}$  inches transversely, and from 3 inches to  $3\frac{1}{2}$  vertically. It contains about a glass of limpid serosity.

The heart is enormously and generally hypertrophied. Opened and washed it weighs, with the origins of the large vessels, nearly two pounds.

*Right heart.* Dilated so as to contain a small orange; the right auricles presents a vermillion tissue and some stout columns. The circumference of the tricuspid orifice is nearly five inches. The valve is thickened, but very well shaped, although a little adherent to the ventricle, towards its middle. The ventricle is dilated proportionally with very stout columns; its wall is about  $\frac{5}{12}$  of an inch thick near the base. The orifice of the pulmonary artery has a circumference of  $3\frac{1}{2}$  inches. The valves are proportionally enlarged, but are very thin and well shaped.

The inter-ventricular partition is nearly one inch thick.

*Left heart.* The left auricle is rather larger than the right. The circumference of its orifice is nearly  $5\frac{1}{4}$  inches. The valve is thickened; almost fibro-cartilaginous, the lamina being so approximated as to give it the appearance of a ribbon; not adherent to the ventricle, but bridled by short tendons which must incommode its play.

The ventricle, the peculiar seat of the hypertrophy, is first remarkable for the strength of its fleshy columns which are short and thick. From the base of one of them there starts a small tendinous band, very thin, which goes to insert itself upon the corresponding side of the ventricle, about 15 lines below the aortic orifice; towards the point of this insertion the endocardium, opaline in this region, presents a kind of

little plate slightly projecting, about two and a half lines in extent, and a half line thick, apparently fibrous. The wall of the ventricle is firm and ten lines in thickness; it does not commence to decrease except in the neighborhood of the point of the heart, which is soft and rounded.

Water being poured into the aorta runs over, showing that the valves are sufficient: they are thickened, however, and notably enlarged, their height is a little more than 10 lines, instead of from 6 to 7 lines, that is to say at least from 3 to 4 lines. The circumference of this orifice is about 3 inches.

Like the right heart the left contains black, soft and unorganized concretions.

Now let us compare the lesions found in the dead body with the principal assertions of our diagnosis.

I had said: *considerable hypertrophy of the heart, 1½ pound*, we found it to weigh nearly 2 pounds. I will not repeat the general reason of this error, but I will say that here a special reason for this under-valuation, was the situation of the point of the heart, in consequence of the ventral effusion, in the fifth intercostal space, and only a little without the nipple.

*Relation and absolute dilatation of the auricles.* I said: the auricles are dilated not only *relatively* as regards their usual capacity with reference to that of the ventricles, but *absolutely*, that is to say, independent of that relation. The hypertrophy is general; for a long time there has existed a stasis of the blood; but we have no signs of mitral or tricuspid insufficiency; then if the blood remains long in the auricles, it is because these, being dilated, have lost their contractility.

The autopsy has confirmed this view, in showing us, it is true, a dilatation no less pronounced of the ventricles than of the auricles themselves, the former having their walls much diminished in thickness and consequently but little force in their contraction. What is a thickness of ten lines towards the base of the left ventricle of a heart weighing nearly two pounds, instead of about  $\frac{3}{4}$  of a pound, when the average thickness of this ventricle is  $7\frac{1}{2}$  lines? This circumstance was doubtless the cause in our patient, of the moderate impulsion of the heart, the smallness of the pulse at the wrist, the imperfect beats, and consequently of the stasis of a part of the blood, all the serous infiltration that we have noted, the distension of the jugulars, the dyspnœa, &c.

*Thickening, deformation and perhaps adherence of the left valves especially.* Why these lesions? Because the two beats are imperfect, as we nowhere have the valvular clacking; we

should consequently suppose the play of the valves to be impeded; and as the absence of souffle and of vibratory tremor excludes the idea of calcareous deposit or of vegetations, then it remained as probable that there were adhesions of the valvular lamina, either with each other or with a neighboring part of the ventricle.

What in effect did we find? A tricuspid valve thickened, enlarged in proportion to the orifice, but adhering towards its middle part; a mitral valve also thickened but bridled by tendons too short, evidently insufficient for an orifice so much enlarged, (5 inches instead of about 4,) permitting in consequence a part of the blood to retrograde in the first time, as is proved by the considerable dilation of the left auricle.

If I am asked how it was that this bicuspid patency was not betrayed by a souffle in the first time, I will say the cause was doubtless in the feebleness with which the sanguineous column was expelled from the dilated left ventricle.

How did we find the arterial orifice? The aortic and especially the pulmonary valves are enlarged, and thus less adapted than in the normal state to the production of clacking.

*Without absolute contraction or vegetation.* I should have added, *or cretaceous deposits.* I have just remarked that the absence of souffle and of tremor put this fact beyond all doubt. The necropsy fully confirmed this point of the diagnosis.

*Perhaps smallness of the pulmonary orifice.* This was a pure supposition based upon a slight whining, distant from the ear, not continued into the carotids; and which I heard at two or three different times between the nipple and the sternum. The autopsy did not verify this hypothesis. I confess however, that I am not yet unconvinced that the pulmonary orifice was not the seat of this feeble and distant, but still, well heard sound, under the influence, not of a fixed lesion, but simply of some momentary and moveable obstacle; such as a small sanguineous concretion afterwards carried away by the torrent of the circulation.

*Inconsiderable effusion into the pericardium.* No one, I think, will object to this on account of the glass of the serum, found after death. For what is this quantity compared to the increased size of the pericardium? The point of the heart which we felt striking under our finger in the fifth intercostal space, prevented our admitting a considerable effusion, which would have been incompatible with the superficial location of this organ.



I say nothing of the pulmonary œdema.

As in the preceding observation, I will terminate this analysis by asking, if the concordance between the diagnosis and the autopsy is not sufficiently complete to prove the value of means of observation by the aid of which we can attain a like practical result?

**OBSERVATION 12.**—*Thickening and deformation of the left valves. Moderate diminution of the aortic orifice.* Joseph Veisin, a porter, aged 53 years, was received Aug. 20th, 1841.

This man, of a very strong constitution, and heretofore a soldier during twenty-five years, had generally, very good health. He had, nevertheless, two attacks of intermittent fever, one in 1808, and the other in 1815, each of which lasted only one month. In 1818, he appeared to have had an affection of the left lung, which continued three months, and finally in 1826, some rheumatismal pains in the arms and especially in the calves of the legs, with fever and abundant sweats. He was then treated at Val-de-Grace by cups and a bath, and left, supposed to be cured, in eight days. He said his health had been very good from that time to the present; but yet remarked that for a year past, his respiration was shorter, and that he had palpitation in ascending. These symptoms have increased during the past month especially, and to them has been added a little turgidness of the inferior extremities, which disappears in the horizontal position. He has kept his bed for three weeks, but not constantly.

*Present condition.* No puffing of the face. Very notable œdema of the feet, legs, and left wrist; no positive signs of ascites, pulse at 76, vibrating, especially the right, which is more developed than the left, in consequence, without doubt, of œdema of the latter.

The pulsation of the right radial are remarked in a very great extent.

A prominence of about six degrees between the left nipple and sternum.

The beatings of the heart are very greatly extended, although the point of that organ, not readily seized at first, beats only in the fifth intercostal space, and rather within than without the nipple. The hand firmly applied to the precordial region perceives a very distinct but profound vibrating tremor. The dullness appears to be only about  $2\frac{1}{2}$  to 3 inches square, but the presence of the lung presents a very exact determination of this matter. In the precordial region, and especially, towards the apex of the heart, we distinguish in the first time



a bruit de souffle, very contracted, and which from time to time almost simulates a whining. The second clacking is quite dull, and in its place we discover a little, but much shorter souffle. Towards the base of the organ, and in approaching the aortic orifice, the first souffle persists, but the second in becoming more pronounced, is accompanied by a very distinct clacking. Setting out from this point, if we follow an oblique line, directed towards the right clavicle, the double souffle becomes yet more marked, and its maximum appears to be about the middle of the track we have indicated. Yet there exists there neither tumors or tremor under the heart. The souffle is propagated into the carotids, these arteries beating strongly and visibly. The same is true of the subclavian; and in the right subclavian we even verify a very notable vibrating tremor. No well-marked distension of the jugular veins. Very strong pulsation of the cœliac trunk, the abdominal aorta, and the crurals, without souffle. Nothing very remarkable about the respiratory apparatus.

If the reader has attentively followed the analysis of our preceding observations, it is impossible, in our opinion, not to read, in the simple expose of the symptoms we have enumerated, and thus to speak, as through a transparent veil, the almost perfect diagnosis of our patient. Let us further seek to extract the principal features of the case, and we will afterwards see if our present diagnosis corresponds with that made at the bedside, and especially with the rigorous testimony of the autopsy.

We have noted, at first, a little partial œdema; there is then a restraint upon the venous circulation. But we have, as in the preceding case, neither general anasarca or ascites, we do not therefrom, expect a sanguineous stasis so pronounced. We cannot specify further until we shall have finished our examination.

Without any doubt, there is general and considerable hypertrophy. I think we cannot deceive ourselves much in estimating the heart at double its normal weight; let us say at from 20 to 22 ounces. But what kind of hypertrophy is it? Is it, as with the preceding patient, in some sort a passive hypertrophy, with dilation and thinning of the walls?

No, certainly, the vibration of the pulse indicates to us an active hypertrophy affecting, very probably, the left ventricle particularly. Let us come, now, to the orifices. This vibration and developement of the pulse excludes all idea of any considerable contraction of the aortic orifice, and any well pro-

nounced insufficiency of the mitral valve itself. Yet, by the application of the hand *on the precordial region*, we perceive a tremor; hence some of the valves must be injured in a high degree.

Now, what shall we say of the auscultation?

Towards the apex of the heart, that is to say in some manner, between the bicuspid and aortic orifices, there is quite a limited souffle in the first time and a much shorter one in the second; there is no clacking. To which orifice do these souffles appertain? Do they both proceed from the aortic orifice insufficiently opened and imperfectly closed? We cannot know it yet; to assure ourselves of it, let us approach our ear to that orifice. The first souffle persists and the second becomes more pronounced; but what is very singular this is now accompanied by a very distinct clacking. Whence comes this singularity? A moment since when the souffle was short there was no clacking, now it is stronger and we have valvular clacking.

The response is easy. It is, without doubt, because the two souffles of the second time, are from different origins; the first was produced at the bicuspid orifice, and however short it may have been, it sufficed, by its proximity, to veil the clacking of the aortic orifice. When we approached this last orifice this clacking was unmasked, and if it is still mingled with a souffle, and especially with a souffle stronger than that which we at first verified, it is probable that this second souffle is owing to a peculiar state of the internal face of the aorta. Let us pursue further our examination and this hypothesis will become a certainty, when in following the track of the ascending aorta, we perceive that it is, in fact, in this region that a double souffle presents its maximum of intensity.

Let us then resume and now say; *Deformation of the two left orifices, the bicuspid valve being, perhaps, insufficient* (no clacking in the first time,) *but in a feeble degree,* (development of the pulse;) *the sigmoid valves not being insufficient* (clacking of the second time); *but the orifice that they close being contracted* (whining of the first time towards the aortic orifice). Let us add: *cretaceous places in the aorta*, and, in fine, to express that which is indubitably announced to us, by the strong pulsation of the arterial system, *hypertrophy of the principal arteries*.

Let us now see, what was our first diagnosis. It was not so perfect. M. Bouillaud being absent, I at first dictated the following at the bedside of the patient.

*General and considerable hypertrophy of the heart.—Thickening and deformation of the left valves.—Hypertrophy of the principal arteries.*

Afterwards determined by the persistence of the whining I added: *the principal lesion is at the aortic orifice, and it consists in a contraction.*

Let us now see what the autopsy will show. It was made on the 24th of September, the patient having died suddenly the day before.

*Autopsy.* No serosity in the pericardium.

The heart globular, rounded and soft at its apex, weighs with the origins of the large vessels, 24 ounces. The hypertrophy affects, especially, the left ventricle. The four cavities contain no remarkable sanguineous concretions.

*Right heart.* The auricle is sensibly dilated, it would contain a very large egg. It is firm and plump, and of a vermillion color on the interior.

The tricuspid valve is a little thicker than natural, but without deformation. The circumference of the orifice is nearly  $4\frac{3}{4}$  inches.

The ventricle presents only a medium capacity, contracted, relating to that of the auricle. Nothing remarkable about its walls, which, even towards the base, are not five lines.

The valves of the pulmonary artery are well shaped; the orifice is nearly  $3\frac{1}{4}$  inches in circumference.

*Left Heart.* The left auricle is dilated so as to be capable of containing a small orange.

The bicuspid orifice, which participates in this dilation, is nearly  $4\frac{3}{4}$  inches in circumference, (instead of  $4\frac{1}{4}$ ); its valve is considerably thickened, almost cartilaginous, roughened on its free edge by small vegetations, yet fleshy without cretaceous matter; the same kind of vegetations on the chordæ tendineæ which terminate in it. The columnæ corneæ are remarkable for their force and magnitude.

The ventricle is less remarkable for its dilation than for the thickness of its walls. These are, in fact, a little more than 15 lines towards the base, and this thickness declines only in an almost insensible manner to the apex. The tissue is firm and of a vermillion red, and recalls by its aspect the most rigorous muscles. The endocardium presents a slightly opaline tint.

The aortic orifice is manifestly contracted both relatively and absolutely; its circumference is  $2\frac{3}{4}$  inches, (5 lines less than the pulmonary orifice, when normally they are about

equal.) Its valves are very notably thickened, especially toward their free border, which is rugous, a little unequal, presenting small vegetations like atheroma, or even, many of them like ostropetrous, small and more sensible to the finger than to the sight. The origin of the aorta also presents some clusters, in plates yet more prominent, of this same matter which grates under the finger when rubbed upon it; we find it as far as the middle of the descending aorta. The aorta is besides remarkable for its thickness, and all the large arteries, particularly in this hypertrophy. One of the crurals opened and placed by the side of a normal crural offers a capacity at least double.

What shall we say of this autopsy, if it is not that its history is found almost perfect in advance, in our analysis of the symptoms? We have seen, moreover in the mean time, the dilation of the auricles, and especially the left one, which is an additional reason for admitting the insufficiency of the bicuspid valve, the corresponding orifice being, besides, notably dilated ( $4\frac{3}{4}$  inches instead of  $4\frac{1}{4}$ ). This dilation of the auricles, the probable but rather secondary course of sanguineous stasis, will, without doubt, explain the limited, but incontestible infiltrations, which we have noted. Yet another feature which is wanting in our analysis, is the existence of those small vegetations, which the vibratory tremor should lead us to suppose; a tremor which in fact, had no other cause unless the aortic orifice might also have contributed to it in a feeble manner. Let us again remark in reference to these small fibrineous concretions, veritable rudiments of calcareous and other concretions, that their deposition on the free border of the bicuspid valve and along the chordæ tendinæ, of this valve, seems to give us the key to their formation.

What, in truth, could be more analogous to the deposition of the fibrin of the blood on broom straws, as it occurs in our laboratories, than these curds, so delicate, agitated without ceasing in the midst of blood, which is so violently shaken by the energetic contractions of the hypertrophied ventricle.

**OBSERVATION 13.**—*Double contraction with bicuspid and perhaps aortic insufficiency.*—*Pericardiac adherencies.*

Lucien Bonde, tailor, aged 27 years, entered on the 2d of September, 1841.

This young man of a medium constitution, rather delicate and of a lymphatic temperament, was attacked at the age of nine or ten, by general acute rheumatism, which has reappeared almost every year, except the last five. At an epoch



which he could not exactly specify, but at least six years before, he began to experience palpitations and suffocations which persisted until the present time, but which put no remarkable restraint upon him until about eight months since. Since then, also, very frequent cough, and latterly there was œdematous tumefactions of the inferior extremities, then of the scrotum, and then of the abdomen. He has kept his bed for three months.

*Present condition.* The patient is seated on his bed. The face is pale and puffed. Considerable œdematous swelling of the feet, legs, thighs, scrotum and penis. Tumefaction of the abdomen, with a little dullness in the lower parts. The extremities are cold. The pulse is at 88, little developed and rather unequal. Three or four degrees of precordial prominence. The pulsation very deep, but extended, accompanied at intervals by a little vibratory tremor beneath the nipple. The apex of the heart strikes the fifth intercostal space, or between that space and the sixth, and the pulsations extend themselves from this point some distance above the nipple and to the neighborhood of the sternum; the precordial dullness being about  $2\frac{1}{2}$  inches square. As to the sounds of the heart, instead of clackings there is only a double souffle, very rude and raspy, much more pronounced in the first time than in the second, of which the sound is almost entirely effaced; this souffle attains its maximum near the inferior hollow of the sternum. At other points we distinguish a *bruit de choc*, very heavy and dull, mingling with the souffle of the first time. No notable souffle in the carotids. These arteries, as well as the subclavian, pulsate with remarkable fervor. The jugulars are a little distended. There is a subcrepitant rale with very numerous bubbles behind, at the base on both sides.

With these signs what lesions shall we diagnosticate? I avow that, at first, this case seems more difficult of perfect analysis than the preceding. Yet, if we consider that there is an absence of valvular clacking, an absence more complete in the first than in the second, we will be brought, already, to admit a double valvular lesion, affecting especially the bicuspid valve. From this complete absence of bicuspid clacking we shall conclude besides, that there is insufficiency of this valve. As to the aortic orifice, we may perhaps hesitate in reference to its insufficiency; but, the almost, if not entire effacement of its valvular clacking will not permit us to overlook a certain degree of alteration of this valve. The vibra-



tory tremor, itself, will also indicate a pronounced alteration of an orifice; but of which? In recollecting the situation of this tremor, below rather than above the nipple, we shall have some reason to attribute it, in preference, to the bicuspid orifice.

Now, such a detailed analysis is not always possible at the bedside of a patient and under inspiration of a first examination. Such, moreover, was not the diagnosis of M. Bouillaud. That clever observer verified, in his turn, what we had noted the day before, and dictated the following:

*Hypertrophy of the heart and of the principal arteries.—Induration and thickening of the left valves, with contraction of the orifices.—Suspicion of adhesions to the pericardium.*

Why this contraction of these orifices? Because this, or a well marked insufficiency, is necessary to explain the coincidence of a pulse so little developed and so unequal, with pulsations, in appearance, so extended; because, without doubt, the contraction appeared, to M. Bouillaud, more probable than the insufficiency, seeing the tone of the souffle, that tone of which the character could be seized only by a practical ear; because in fine, the vibratory tremor of itself, gave sufficient grounds for the diagnosis.

Finally, why suspect pericardial adhesions? Because as M. Bouillaud caused us to observe, there were pulsations, very deep, extended, and restrained; because we did not find, under the hand or ear, that open exaggerated play, that free impulsion of a simply hypertrophied heart. I avow that nothing less than all the habitual and consummate experience of our modern Corvisart, could have enabled one to seize these shades of diagnosis, which were, nevertheless, expressed only under the form of suspicions.

Again the verification of the autopsy was not long delayed. On the evening of the 9th of September, the patient died in a state of orthopny and general depression.

*Autopsy.* The pericardium contains five or six tablespoonfuls of a reddish liquid, as though bloody. The opening of this cavity on its anterior face is accomplished only with difficulty in consequence of the intimate adhesions and very short filaments, which unite the pericardium to the heart, especially to its anterior and left lateral faces, adhesions which it is necessary to dissect in order to expose the heart naked. In the remainder of its extent, and especially the right ventricular face, the heart presents many thick whitish plates, which we can detach only with difficulty.

The heart is considerably and generally hypertrophied. This hypertrophy effects, especially, the left ventricle and the two auricles. In the interior it contains only some soft and blackish concretions. Washed, it weighs, after some moments drying, about  $1\frac{1}{4}$  pounds.

*Right Heart.* The auricle enormously dilated, might contain a very large orange. It is firm, fleshy and very red. The circumference of the tricuspid orifice is  $4\frac{1}{2}$  inches (normal). Its valve is greatly thickened, fibro-cartilaginous, especially towards its free border, and adherences to the ventricle by the extremity nearest to the pulmonary artery. The tendons which are inserted therein are stout, firm and join to some very robust and thick columns. Yet the right ventricle is but little dilated, proportionally, and its maximum thickness, which corresponds with its base, is only 5 lines.

The valves of the pulmonary artery are well shaped. The orifice is  $3\frac{1}{8}$  inches in circumference.

*Left Heart.* There is nothing very notable about the calibre or the internal face of the aorta. The valves, sensibly thickened, preserve a little transparency towards their medium part, but their free border is transformed into a cartilaginous excrescence, creaking under the scalpel, hard to the finger, without ossification or notable deformation, unless these valves adhere together by their adjacent border in such a manner as to contract the opening of that orifice, which in preserving these adhesions offers a circumference of only  $2\frac{1}{2}$  inches.

The left ventricle is dilated and might lodge a large egg.—Its walls are thickened, firm and red; are nearly ten lines thick almost as far as the apex, which is quite soft and rounded. The columnæ corneæ are generally stout, especially those which terminate by their tendons in the bicuspid valve.

This valve, not having been cut, presents in its centre a rather oval opening nearly seven lines in extent, which, as a sort of vent hole, allows the light to penetrate to the bottom of the ventricle. This opening is, moreover, entirely circumscribed by a cluster of cretaceous matter, forming a circular series of concretions or nuclei, of which many have the volume of large peas. They are besides, unequal, very hard, rough and almost perfectly replace the valve, thus become unrecognizable, and some of them descend even like stalactites in the thickness of the tendons which are there fixed. The circumference of the auriculo-ventricular orifice, measured

without opening it, is  $3\frac{1}{2}$  to  $3\frac{3}{4}$  inches (instead of  $4\frac{1}{4}$ ). The corresponding auricle is large enough, but rather less than the right. It is nevertheless very firm, fleshy and red.

The two lungs, and especially the left, are engaged posteriorly.

This autopsy has little need of comment. Save the hypertrophy of the arteries, which I do not find noted, and that perhaps because we omitted to seek it, the diagnosis of M. Bouillaud, is confirmed in every point; *adherences of the pericardium, contractions of the left orifices*. Let us remark, that the insufficiency of the bicuspid and the predominance of lesion of that valve are also verified. The signs which had fixed our attention on this double circumstance did not then deceive us.

I now no longer doubt, that the adherences of the aortic valvular laminæ to each other, whence resulted, in some sort, the corrugation and contraction of the corresponding orifice, may have had no other result than a certain insufficiency of these valves; and that our feeble clacking of the second sound may have been produced much less by these valves than by those more remote, but normal, of the pulmonary artery.

But shall we say nothing of the tricuspid orifice? Its circumference, which, though of the same size as in the normal state, was, in fact relatively contracted, diminished still more by the adherence of a part of its valve to the ventricle, did it not find itself by this double cause morbidly contracted, imperfectly closed with patency, which we should remember to explain, both the dilation of the right auricle and the obstacle to the venous circulation, the cause of the serous infiltrations noted above? For my part I am inclined to think so. We may, moreover, ask if it was not the tricuspid lesion which gave rise to the souffle we heard in the inferior hollow of the sternum, a region frequently indicated, and first by Laennec, as the seat of the morbid sound of the right heart. This is a question which I am content for the moment with having suggested.

Thus, then, to resume our principal morbid phenomena, *vibratory tremor*; cretaceous state of the bicuspid orifice.

*Souffle of the first time*: aortic narrowness, bicuspid insufficiency, and perhaps tricuspid, the two last orifices being incapable of producing clacking.

*Souffle of the second time*: bicuspid and tricuspid narrowness, and a little aortic insufficiency.

*A little clacking in the second time*: imperfect play of the aortic valves, the clacking of the pulmonary being masked by the souffle.

It now remains for me to obviate an objection. If the aortic orifice contributed to the production of the first souffle, why did the latter not propagate itself into the carotids? Because, without doubt, the sanguineous column, divided in the left ventricle between the bicuspid orifice, remaining open, and the aortic orifice, did not beat in sufficient quantity against this last orifice to cause the souffle resulting from this friction, to propagate itself as far as the carotids. This is proven by the feebleness of the pulse. And now if one should oppose to this explication, the forcible beatings of the carotids and subclavians, I would respond that this force is not always in proportion to the column of blood which traverses them; witness the localized partial pulsations of the cœliac trunk and of the carotids themselves which often occur in chlorotic persons, and which have sometimes imposed themselves as aneurismal pulsations, or which we have signalized in many cases of notable disturbance of innervation, and in the cholera itself. (See the memoir of MM. Serres and Norat on Cholera.)

OBSERVATION 14.—*Bicuspid contraction and insufficiency.*  
—*Thickening of the aortic valves.*—*Tricuspid insufficiency.*  
—*Pericardial adherences.*

Joseph Viard, varnisher, aged 22 years, was received the 4th of October, 1840.

This young man, of a lymphatic temperament, enjoyed good health until 18 years old, at which time he had general acute articular rheumatism, which lasted three months, and for which he was treated in this hospital. During that attack there were palpitations and very severe precordial pain. He said he recollected that in being ausculted, a bruit de souffle was heard. These last accidents detained him a month longer in the hospital, where they were met by cups and opiate cataplasms. Finally the patient was discharged cured, feeling no more, he says, either palpitations or suffocation.—At the end of two years another attack of rheumatism occurred, with yet more severe palpitations. This time it was only after a sojourn of six months that the patient was able to leave the hospital, and ever since then he remained subject to palpitations, which, for eight months, have prevented him from returning to his occupation, and to which has been added, within eight days, infiltration of the inferior extremities, the genitals and the abdomen.



*Present condition.*—The patient is seated, his legs suspended out of bed; they are infiltrated, especially in their inferior third, as well as the scrotum and skin of the penis. The abdominal cavity distended by a considerable effusion, gives a perfectly dull sound as far as about ten lines above the umbilicus. The face is also notably puffed, it is moreover pale and anemic; the lips are rather violet, and the extremities cold.

The pulse is at 96, small but very regular. No precordial prominence or vibratory tremor, but the impulsion is very strong and extended. The apex of the heart beats in the sixth intercostal space, a little without the nipple. The vertical dullness is about  $3\frac{1}{2}$  inches, and more than  $3\frac{1}{2}$  transversely. We hear in all the precordial region, but especially in that of the left cavities, a double souffle, rude, raspy, following the valvular sounds, prolonged, especially in the second time, and accompanied by a mixture of auriculo-metallic tinkling and a heavy, dull sound, the double result of the shock of the heart against the pectoral walls. We note, in fine, on the part of the respiratory apparatus, a subcrepitant rale, well formed behind in both lungs, especially the right.

The next day M. Bouillaud added to the preceding, that in approaching the right subclavicular region, second sound is relieved, and it is there heard very distinctly giving a slight parchment tone.

This addition is important, for it puts us in a condition to diagnosticate the disease in a manner probably complete and precise. We have a double souffle, does this alone indicate a double lesion? No! without doubt, or, if it is double, it is capable of predominating at one of the two orifices. Which is that orifice? On one side we have no very distinct souffle in the carotids; on the other, in receding from the heart we reach a point, where is found the clacking of the second time modified only in its tone. There it is the bicuspid orifice which is especially injured, and of which the valve is probably insufficient, and the diameter contracted, the aortic valves being thickened but sufficient. Fortified by the results of our anterior observations, we may add, that the extent of the serous infiltrations, induces us to admit, besides, a dilation of the right auricle (without detriment to the left) with dilation and perhaps insufficiency of the tricuspid orifice.

M. Bouillaud contented himself the first day in diagnostivating: *a general hypertrophy of this heart, with thickening*



and deformation of the left valves. Some days after, he dictated the following:

“The raspy sound, which absorbs the valvular clacking, is very rude, as if contracted in the left cavities, where we rather feel a shock, than distinguish sounds; but towards the inferior part of the sternum, the double raspy souffle becomes more pronounced and aniple; it seems as though the heart were restrained in the pericardium which envelops it.”

On the 22d of November, the patient succumbed. Having been prevented, by indisposition, from assisting at the autopsy, it was dictated by M. Bouillaud.

*Autopsy.*—The parietal fold of the pericardium adheres in all its extent to the cardiac, a circumstance which explains the restraint in the movements of the heart, which was manifested in the living subject. This adherence is very intimate and strong by means of very close cellular tissue.

The heart is gorged with clots. Hypertrophy, with dilatation of the right ventricle and auricle; the auricular columns are double their normal size. The thickened tricuspid valve forms a kind of narrow ribbon, fibro-cartilaginous, incapable of closing the orifice, which is a third larger than in the ordinary state. The pulmonary valves are sound.

Water poured into the aorta rises; the aortic valves preserving nearly their normal form, are only a little shrunken and opaque in consequence of their fibro-cartilaginous thickening. They are four times their normal thickness, and are a little red on their free border. The orifice is not notably contracted.

The left ventricle is of a volume and weight at least double that of its normal state, with dilatation of its cavity; its walls have a thickness of about 1 inch.

The two laminæ of the bicuspid valve are united at their commissure in such a manner as to form a fibro-cartilaginous ring, the lips very thick, analogous to those of the glottis, with contraction of the corresponding orifices, which very readily admits the end of the index-finger.

The internal membrane of the left auricle is thickened in all its extent, wrinkled and a little slagreened. The muscular bed is about  $2\frac{1}{2}$  lines thick.

In both lungs we have only to note a serous or sanguineous congestion toward the dependent parts.

It is apparent that the reflections, which the preceeding observation presented to me, are in a great part applicable to this. I therefore refer the reader to it. And in fact, we have

in this again pericardial adherences diagnosticated, or at least suspected; the contraction and bicuspid insufficiency; tricuspid insufficiency, with which, by the by, we will again see coinciding, a souffle towards the inferior hollow of the sternum; in fine, an aortic lesion without carotidian souffle. As in the preceding observation, I attribute this last anomaly to the reflux of the blood by the bicuspid orifice; I doubt not, that without this circumstance the aortic souffle would be propagated to the carotids.

**OBSERVATION 15.**—*Bicuspid and tricuspid contraction. Bicuspid insufficiency. Slight aortic thickening.*

Julian Choisy, keeper of a coffeehouse, aged 49 years, entered our service on the 29th of May, 1841.

Of a medium constitution and lymphatic temperament; at the age of 9 he was affected by a general articular rheumatism which lasted five or six months. At 21 there was a new attack which had the same duration. In 1826 a third attack occurred which lasted many months. Seven years ago, for the first time he experienced suffocation and palpitations, which required a great many applications of leeches to the epigastrium and several bleedings. Four years ago there was a new general rheumatism of five months duration. Since then there have been palpitations from time to time, more intense during the last five months, with dimness of sight and stupor; some cough since one month, and for a few days past a little œdema of the feet.

*Present condition.* Face a little pale and puffed; no ascites or œdema of the inferior extremities; heat moderate. The pulse at 80, rather unequal, quite regular, of medium developement and not vibrating.

The precordial region presents a prominency of 4 to 5 degrees by the *cystometre*. The apex of the heart beats directly below the nipple in the sixth intercostal space. We also remark some pulsations in the inferior sternal hollow. The impulsion of the heart is of a medium force, but of very notable extent, the precordial dulness being about 3 inches square. No vibrating tremor. The first sound of the heart is perfectly replaced by a raspy souffle, very contracted, recalling to mind the whining. This souffle extends into all the precordial region, but above all into the region of the left cavities, and especially the auriculo-ventricular orifice. In proportion as we ascend along the aorta, this souffle decreases and even disappears very promptly, giving place to a heavy and dull sound. It is not propagated into the carotids. As to the second sound,

distinct in the precordial region itself, it consists, instead of clacking, of a very hard and heavy sound without soufflé. The jugulars are a little distended. The dyspnœa is moderated by repose. We note, on the part of the respiratory apparatus, only a little sub-crepitant rale disseminated behind, especially to the left, and some small, dry creaking, probably pleural, before and above the left nipple.

The patient reports that a month since his habitual dyspnœa become so strong as to require a bleeding, and that it was accompanied by a very acute pain.

Let us now analyze these different symptoms, and we shall see to what diagnosis they will conduct us. Do we find at first, in the habitual exterior, an indication of any very considerable, obstacle to the venous circulation? We have the puffing of the face, a little distention of the jugulars, and a slight pulmonary œdema; but no ascites or infiltrations of the limbs. We ought then to suppose a certain degree of restraint to the circulation of the right heart, and as a consequence a slight dilatation of the auricle of that side; but not yet those considerable dilatations which we have seen bound, for example, to tricuspid insufficiency; rather then a little narrowness, at least relative to that orifice. The pulsation that we noted in the inferior sternal hollow, puts us in a position to think that the right ventricle participates in the hypertrophy, otherwise considerable, of the entire heart.

Let us now come to the left cavities. In the region which corresponds to them, especially in that of the bicuspid orifice, that which strikes us of course is a very rude and contracted soufflé, predominating in the first time, not even encroaching sufficiently on the second time, to prevent the appreciation of the valvular clacking, which, moreover, becomes more pronounced as we approach nearer to the aortic region, but with a very hard and heavy tone.

Can we here overlook a bicuspid insufficiency with a certain degree of contraction of that orifice at the same time, and a certain thickening of the aortic valves which does not prevent this adjustment, (for they are not insufficient,) but which obscures their clacking; a thickening which may well be supposed to have some influence in the production of the soufflé of the first sound; but this influence is necessarily limited, since the soufflé does not extend to the carotids, the orifice being, moreover, sufficiently open to permit the column of blood feebly decreased by the bicuspid insufficiency, to produce pul-

sations at the wrist, not vibrating and rather unequal, but still sufficiently developed ?

But what is the dull, dead sound along the aorta, which is by degrees disengaged in proportion as the souffle of the first time decreases ? It is the imperfect clacking of the bicuspid valve, which, insufficient to some extent, has not lost all its play and to which moreover is associated the more distant, and doubtless somewhat modified, clacking of the tricuspid valve. Besides, we have no vibratory tremor ; nor have we either vegetations or a cretaceous state of the orifices, nor of the aorta.

Let us give now the diagnosis dictated at the bedside of the patient by M. Bouillaud.

*General hypertrophy of the heart, (18 ounces) thickening, induration and deformation of the left valves, especially of the bicuspid, with the contraction of the corresponding orifice.*

The patient died the 15th of June without presenting any new local symptoms.

*Autopsy.* The cardiac layer of pericardium presents several plates, particularly three, one towards the base of the left ventricle, another towards the base of the right, and the third towards the summit of the left ventricle. These three plates are in more or less bold relief.

The heart is generally and considerable hypertrophied, the morbid developement affecting especially the left heart.

All its cavities contain sanguineous concretions, of which several were fibrinous before death. Disembarrassed of these clots, and after having macerated in water for near five hours, the heart weighed 22 ounces. The water with which it is impregnated evidently adds to its weight.

*Right heart.* The auricle is sensibly dilated, sufficiently firm and vermillion in its tissue. The circumference of the tricuspid orifice is only 4 inches, instead of  $4\frac{1}{2}$ . Its valve is thickened, and, on the side nearest the pulmonary artery, it is slightly adherent to the corresponding part of the ventricle. This is but little dilated, either absolutely or proportionally.

The maximum thickness of its walls is 5 lines. The pulmonary valves are well shaped, only a little stronger than normal. The corresponding orifice is  $3\frac{1}{4}$  inches in circumference.

*Left heart.* Water poured into the aorta immediately overflows. The calibre of the vessel offered nothing extraordinary. We remarked only on its internal face and towards its origin some slight rudiments of cretaceous matter, not as

yet protruding notably. The sigmoid valves perfectly formed, are firm and sensibly thickened without having completely lost their transparency. The aortic orifice is 3 inches in circumference.

The left ventricle, the essential seat of the hypertrophy, is sensibly enlarged in its cavity, which could hold an egg, and thickened in its walls, which are 12 lines thick at the base and 10 lines towards the summit, which is softened and rounded. The columnæ-corneæ are proportionally robust.

The auricle is likewise sufficiently larger, its tissue is vermillion and fleshy. The circumference of its orifice is  $3\frac{3}{4}$  inches, (instead of  $4\frac{1}{4}$ ). The very much thickened valve, more than all the others, fibro-cartilaginous, creaking under the scalpel, adheres strongly to the ventricle by a part of the internal face of its free border, and especially in a third of its extent; it is then insufficient. The two columns which are inserted there, are strong and terminated by voluminous tendons.

The two lungs present a little serous engorgement at their posterior parts.

First let us remark, that I was deceived in taking for a pleural friction that which was evidently due to the patches found on the pericardium. This error had been easily avoided if the dyspnœa of the patient, though moderate, did not prevent us from obtaining a complete and slightly prolonged suspension of respiration, and especially if the morbid sound in question, partly masked by the soufflé, had itself been more persisting than it was; this circumstance owing perhaps to the inequality of the ventricular contractions, was evidenced by the inequality of the pulse.

Now with exception of this inexact appreciation of an accessory phenomenon, and relatively to what concerns the orifices of the heart, has not the autopsy, in all points, confirmed the diagnosis?

We had here, too, a tricuspid lesion, and we did not notice a soufflé in the inferior sternal depression; this is a negative reflection to be put in opposition with the remarks that two of our anterior facts have suggested.

*OBSERVATION 16.—Contraction of the two orifices. Bicuspid vegetations, cretaceous concretions on the aortic valves.*

Auguste Camuset, aged 16, was received, into our service on the 21st of May, 1841.

This young patient, of a delicate constitution and lymphatic temperament, has heretofore habitually enjoyed good



health. A year ago he had general articular rheumatism, which detained him in bed two or three months, and since then he has remained subject to articular pains, from time to time; and especially for three months past he has experienced, palpitations, dyspnœa and many symptoms of cerebral congestion. Finally, for some time there has been turgidness of the inferior extremities, extending even as far as the trunk.

*Present condition.* A little puffing of the face. Infiltration of the feet and the inferior portions of both legs. Œdematous infiltration of the coats of the scrotum, with general swelling and transparency, which at first sight simulates hydrocele. The skin on the trunk rather warm, being  $38^{\circ}$  (centigrade) on the abdomen. The pulse is 136, very small, but quite distinct, a little vibrating and regular. General præcordial prominence imperfectly circumscribed. We remark some pulsation in this region, which are propagated as far as to the inferior hollow of the sternum; those of the apex of the heart raising the sixth intercostal space a little without the nipple. These pulsations are very strong, but especially remarkable for their extent, accompanied by a sort of heavy and distant tremor, which is prolonged in the track of the ascending aorta. The dullness over the heart is about  $2\frac{3}{4}$  to 3 inches square.

In applying the ear to the præcordial region we are struck, and in some manner astonished, at the truly exceptional intensity of a souffle, at the same time raspy and analogous to a jet of vapor, a double souffle, extending itself equally into the right and left breast, reaching as far as to a point under the left clavicle, and ceasing to mark the second sound only under the right clavicle. It is, in fact, only at that point that we hear the clacking of the second time, a hard, dry, essentially parchment clacking analogous to a very vigorous fillip. The souffle of the first time which we again distinguish in this region is propagated, but feebly, into the carotids. These arteries, and especially the right subclavian beat with force, in a manner sensible to the eye. The jugulars are manifestly distended. No notable souffle in the cœliac trunk nor in the abdominal aorta or crurals; the sound of these last also reminds one of the sensation of a very distinct fillip. The respiration is restrained, a little plaintive, from 44 to 48. Nothing else to note in the sounds but a little disseminated subcrepitant rale.

At the visit of the next day, M. Bouillaud also verified all the preceding signs. He noted farther that the præcordial souffle

extended to the distance of 2 or  $2\frac{3}{4}$  inches from the pectoral walls, under the form of a slight whining, which, when we applied the ear, was effaced by the rudeness of the bruit de souffle. As on the evening before the second sound disengaged itself only towards the right clavicle, under the form of a parchment clacking, a little veiled, the first remaining masked by the souffle. Behind the chest, on both sides, the double souffle is heard with force, completely covering the two normal clackings.

From all the signs we have enumerated, let us try to form a diagnosis.

As in the preceding observation we have at first some reason to admit a stasis of the venous blood and a medium dilation of the left auricle. The left orifices are evidently both injured. A souffle so intense, and which remains double and without mixture of clacking in so great an extent, excludes the idea of an unique lesion. Yet a little clacking is heard in the second time under the right clavicle and the souffle of the first time propagates itself only feebly, into the carotids. Then, the principal seat of this enormous souffle is not in the aortic orifice, of which it seems the valves, without doubt thickened and like parchment, have not lost all clacking. We willingly admit, then, that if our two valves are insufficient, at least the bicuspid is more so than the aortic, since we find some vestiges of aortic clacking, and that of the bicuspid is nowhere encountered.

But let us remark, that in placing ourselves in certain conditions, our souffle presents a certain degree of contraction and resembles whining; then, without doubt, one of our orifices is much contracted. Which is it? We have admitted that the insufficiency, that is to say, the enlargement predominates at the bicuspid orifice; then it is to the aortic that we must especially attribute the contraction.

Finally we have a profound and as it were distant vibratory tremor, but also extending itself along the ascending aorta. This is a reason for suspecting vegetations, or a cretaceous state perhaps of both valves, but especially of the aortic.

The diagnostic of M. Bouillaud was not less detailed. It is as follows, dictated at the bedside of the patient.

*General hypertrophy of the heart. Thickening, hypertrophy and probably vegetations of the left valves. Contraction of the orifices, especially relatively to the volume of the heart and to the capacity of its left cavities. Dilatation of the right auriculo-ventricular orifice, and perhaps insufficiency of its valve.*

The following days the signs above noted persisted. On the 24th of May, the souffle imitated the chirping of a young chicken, mingled moreover with a raspy souffle and profound vibratory tremor.

On the 27th, the tremor was yet more pronounced, the pulse vibrating, but at the same time, very small and rather soft. The infiltration of the extremities, scrotum and abdomen made continual progress; the oppression was extreme; finally, on the 31st, the patient died.

*Autopsy.* The heart, freed from some clots, weighed only 10 ounces. (The heat being very elevated, one may suppose that this low figure was owing to the transudation of the fluids.) It is, nevertheless, generally hypertrophied, especially in its left cavities.

The right auricle is dilated; its valve is a little thickened, and its circumference is  $4\frac{3}{4}$  inches. The maximum thickness of the walls of the right ventricle is rather more than  $2\frac{1}{2}$  lines.

On the contrary, the left ventricle is notably dilated, it might contain a small hen egg. Its tissue is firm and resisting. Its walls have a maximum thickness of rather more than 10 lines. Its columnæ corneæ are strong in proportion.

The bicuspid orifice presents an extent of  $3\frac{3}{4}$  inches.

The valve is of a medium thickness, but we remark on its surface, and over an extent as large as a franc, a group of small vegetations, yet fibrous and strongly adherent; others, also numerous, are fixed on the tendinous cords which terminate in that valve.

The aortic orifice is small and very narrow, both absolutely and relatively to the capacity of the ventricle. Its valves are notably thickened. The two nearest the auricle give insertion to a globular mass of the volume of a large filbert, evidently composed of fibrous granulations, which are successively deposited around a central nucleus, as by a sort of organic crystalization. This mass, in which we find some grains as hard as gravel, makes a prominence of about 5 lines in the ventricle, being deposited like grafts on the ventricular face of the valves and upon their free border. There remains of these only some vestiges; they are crisped, retracted and confounded, except at their adherent border, which the mass indicated. On the aortic side we distinguish their free face, that is to say, that upon which the fibro-calcareous mass is not inserted.

I ask of all readers in good faith, save that curious aortic

concretion, which could not have been otherwise indicated but under the name of vegetations, is it possible for a diagnostic to approach more nearly a cadaveric description? Will it be said that the tricuspid orifice was not dilated, because its circumference was only  $4\frac{3}{4}$  inches, i. e.,  $2\frac{1}{2}$  lines more than the normal? But I wish it observed that the question here is of a heart of 16 years; of a heart, all hypertrophied as it is, weighing not  $1\frac{1}{4}$  or  $1\frac{1}{2}$  pounds, but 10 ounces; of a heart, in fine, of which the right cavities are not the predominant seat of the hypertrophy; I wish it remarked that the normal relationship between the two auricular orifices, being represented by one, the difference here is  $2\frac{1}{2}$ , so that the tricuspid is much dilated, or the bicuspid greatly contracted. I know that the tricuspid valve could not well have been insufficient, and yet I would not have been willing to answer for it; but let us remark, that this peculiarity was announced only under doubt. As to that which concerns the left orifices, their narrowness is too palpable to require remark.

And now that all the lesions of the heart are known to us, is it necessary to return to the morbid sounds that it presented, to analyze again their mechanism and the value of the signs? Who does not perceive that our double souffle had its cause at the same time in both the left orifices, that is to say, in the collision of the sanguineous wave against the bicuspid vegetations and the aortic concretions; that the cause of the whining was also in the narrowness of the bicuspid orifice, incompletely closed, perhaps, by a valve whose play was restrained by vegetations, and especially in the smallness of the passage which the state of the deformation of the aortic valves left to the sanguineous column; that the souffle of the first time, propagated itself only feebly into the carotids, because the state of the aortic valve was only one of the elements of this souffle, to the production of which, without doubt, the bicuspid orifice contributed, and perhaps even the insufficiency of the tricuspid valve; that our souffle was equal in both times, because we had, at the two orifices, two anatomical states almost identical, vegetations of one and a cretaceous state of the other, the first souffle having in part for its cause, aortic contraction, bicuspid and perhaps tricuspid insufficiency, and on the other hand, the second souffle being produced by the very probable insufficiency, at least partial, of the aortic valves, and especially by the onset of the blood against the bicuspid vegetations? Who does



not see, in fine, that if very far from the heart, we hear a little valvular clacking in the second time, that it appertains in a feeble manner to the aortic valves, perhaps yet a little moveable, and especially to the normal valves of the pulmonary artery.

But we have said enough on this observation, which, although rather more complex in appearance than any of the others, is not less susceptible of a rigorous and positive analysis.

**OBSERVATION 17.** — *Bicuspid contraction.*—*Aortic insufficiency.*

A patient aged 48 years, was received into our wards on the 8th of December, 1841. He was sent to us as having been attacked at home, about twelve days before, with a pneumonia fruitlessly treated by repeated bleedings. The examination of the patient did not fully confirm this diagnosis, and what, for us, was most positive was the fatal result of a very active treatment (5 bleedings in four days, then 25 leeches and two blisters) directed against a doubtful disease. And in effect, the patient, of a constitution otherwise deteriorated, presented to us all the characters of the most complete anemia, a general palor, extreme feebleness, respiration doubtless full rapid, difficult, plaintive, but everywhere the resonance was good, and nothing else notable in the way of morbid bruits, than some disseminated rales more bronchial than vesicular. As to his anterior diseases, they seemed to us to consist specially in an intermittent fever of near one year's duration, with which he was attacked in 1814, and a disease of the chest in 1821.

After the exploration of the respiratory apparatus, the results of which I have just given, we examined the circulatory system, I found the following:

The pulse 88, languid, feeble, scarcely striking the finger, unequal, and from time to time becoming almost imperceptible. The precordial dullness is fully one third more extended than normally. No vibratory tremor. The impulsion of the heart is deep seated, it beats scarcely perceptible to the touch. Consequently, the sounds themselves are very feeble, difficult to seize, the patient being able, but with difficulty, to suspend, or even to moderate his respiration. However, I distinguished at the time of his entry, and M. Bouillaud, on the following morning, with me, a souffle, double in the aortic region, unique in the left auriculo-ventricular region, existing there particularly in the second time, and slightly file-



like. At this time there was no bruit de diable (humming-top sound); but a distinct propagation of the aortic souffle.

The diagnosis of this affection of the heart is so simple that I hesitate to join it in advance.

Preoccupied by the respiratory apparatus of the patient, and justly indignant at seeing to some extent compromised his method of repeated bleedings, and a very precious method for the skillful physician, but a very dangerous one in inexperienced hands, M. Bouillaud was content to dictate relatively to the heart, the following diagnosis: *organic affection of the heart, and especially of the left valves.*

For my part, as the nature of my lessons each evening obliged me to be more minute, I added the following to the diagnosis: *Deformation of the two orifices, and particularly of the aortic. Insufficiency of the aortic valves; sufficiency of the bicuspid.*

In a few words I can give the reason of this diagnosis.—We hear a souffle in the region of the aortic orifice, during the first time, and it is continued into the carotids; so the aortic orifice is incompletely open, but this souffle is likewise reproduced during the second time; then the orifice is besides incompletely closed. We hear a souffle in the bicuspid region, which souffle, single at this point, exists only during the second time; then this orifice, though it may be sufficiently closed, can be but incompletely opened. From its sound like filing, I ought to concluded that this orifice is ever decidedly contracted, and if in place of *deformation* I had said *bicuspid contraction*, my diagnosis would have perfectly agreed with the lesions found after death.

The autopsy which shows us this, took place December 12; four days after his arrival at the hospital.

*Autopsy.*—The lungs offer nothing notable except some old adhesions, a posterior engorgement, a little redness of the bronchial tubes, and an emphysematous state to the extent of 2 or 2½ inches square of the layer of the left lung nearest the precordial region. The heart is distended with clots, partly of recent formation, and in part formed anterior to death.—Disembarrassed of these clots, it weighs 21 ounces.

*Right heart.*—There is nothing remarkable in the pulmonary artery, its valves are healthy, and the circumference of its orifice is 3 inches.

The ventricle is a little dilated, its walls are scarcely 2½ lines thick. The auricle on the contrary, is notably dilated

—its valve is sufficiently thin; the circumference of its orifice is 5 inches.

*Left heart.*—The auricle is so much dilated that it could, like the other, hold a middle sized orange. The valve is sensibly thickened, but free, without adhesions, and well formed. The circumference of its orifice is only 4 inches. The walls of the ventricle, towards the base, are 10 lines thick. Besides the ventricular cavity is considerably augmented.—Largely open, this ventricle presents transversely an extent of near 8 inches (20 centemittres).

The aorta is notably dilated in its arch. It presents on its interior a rough, unequal surface, studded with chalky deposits which are prolonged even into the descending aorta. As to its valves they are thick, almost fibrous, adherent by their corresponding edges, puffed in their free edges, and finally manifestly insufficient, for water poured into the aorta immediately penetrates into the ventricle. The circumference of the aortic orifice is moreover  $3\frac{1}{4}$  inches.

This autopsy fully confirms our diagnosis so far as concerns the left orifices. But it likewise furnishes two elements omitted by us, on account of the absence of the signs which we have hitherto seen to announce them, I mean the notable dilatation of the right auricle and of the corresponding orifice on the one part; on the other, the chalky state of the aorta. We conceive without difficulty that this last lesion could not have been expressed, either by a vibratory tremor, or by the intensity and rudeness of a rasping souffle prolonging itself along the tract of the aorta, for these signs pre-suppose a vigor in the contractions of the left ventricle, not compatible with the feebleness and anæmia of our subject. But how happens it that the dilatation of the right auricle had not brought on any of those serous infiltration offered by the preceding observations? I confess that this is more difficult to explain. We can conceive, however, that these two morbid elements revealed by the autopsy, have nothing incompatible with the stethoscopic signs noted by us during the life of the patient. It makes only two additional conditions to add to those before mentioned as the cause of the souffles both of first and second time.

The following observation is another one of these that I collected under the dictation of M. Bouillaud, at the epoch when I commenced to follow the visits of this professor, a source fertile in clinical instruction of every nature, especially upon the subject which now occupies us.

**OBSERVATION 18.**—*Double insufficiency and contraction.*—*Cretaceous state of the bicuspid.*—*Adhesions of the pericardium.*

Jean Hans, a cabinet-maker, aged 28 years, was received December 13th, 1836.

This patient is of a feeble constitution, and of a sanguineo-lymphatic temperament. I find that amongst other diseases he has had two attacks of rheumatism, and a bronchial affection accompanied by spitting of blood. At the time of his entry, he was as follows:

Slight precordial prominence. Dullness over the heart for more than 5 inches, both vertically and transversely. A double souffle accompanies the two sounds, but especially the last. The first sound is a bruit de rape (sound of a rasp) sufficiently short, and particularly audible in the region of the left auriculo-ventricular orifice. The second is a prolonged bruit de souffle, analagous to a jet of steam as it escapes from a safety valve; it corresponds especially to the sternal region of the aorta. In spite of the general feebleness, pulse is a little wirey, it is regular, and of mediocre development.—There is no infiltration of the legs.

M. Bouillaud dictated the following diagnosis:

*Enormous hypertrophy of the heart, with thickening and insufficiency of the left valves.*

On the 28th of December, we discovered a vibratory tremor well marked below the breast, and a peculiar sound like that caused by a curry-comb, which made M. Bouillaud suppose that the pericardium was diseased.

Having gone out on the 6th of January in some measure relieved, the patient returned February 15th, attacked with an epidemic bronchitis, with some fever. The bruits de souffle of the heart are the same as heretofore. M. Bouillaud is still of the opinion that a friction of the pericardium is mingled with it.

Nothing essential was observed during the following weeks, except, at the commencement of March, a little puffings of the face, a pulse still small, disproportioned to the beats of the heart, but very distinct, and offering of itself account a little vibratory tremor. Towards the aortic orifice and the tract of the aorta, the souffle of the first time is mingled with a stifled clacking; the second souffle is unmingled and as it were by aspiration.

May 19th, we discovered a swelling of the legs, commencing to invade the thighs. The two sounds of the heart resemble a double *bruit de scie* (sound of a saw.)

22d, a double souffle is heard in the bicuspid region, and there completely covers the valvular clacking. In the region of the aortic orifice, and, on mounting towards the aorta, the souffle still predominates during the second time, the first being mixed with a little clacking.

M. Bouillaud thinks that there is *a considerable contraction of the left auriculo-ventricular orifice and insufficiency of the aortic valves.*

The following days an intense oppression set up, then puffiness of the cheeks and lips, œdema of the superior extremities, diarrhœa, and finally on the 29th of May, death supervened.

*Autopsy* made by M. Bouillaud.—The pericardium is uncovered to the extent of 6 inches transversely, and rather more than  $6\frac{1}{2}$  vertically. The parietal layer adheres throughout its whole extent to the cardiac, by fully organized and dense cellular tissue. The heart, disembarrassed of the clots which distended it, and washed, weighed 27 ounces. All of its cavities are hypertrophied and dilated.

The orifice of the pulmonary artery is free, a little dilated, its circumference is  $3\frac{3}{4}$  inches. Its valves are thickened, especially their free edges, and at their base they are opaline. The right ventricular cavity is one third larger than usual. The same is true of the corresponding auricle, the walls of which are towards the base of the ventricle 5 lines thick (instead of 1 or  $1\frac{1}{2}$  lines). The tricuspid valve is well formed, but of a triple thickness and fibro-cartilaginous; the circumference of the corresponding orifice is  $5\frac{1}{2}$  inches; neither the tricuspid nor pulmonary valve is insufficient.

Water poured into the aorta partly overflows, but some of it penetrates into the ventricle. The aortic orifice is  $2\frac{3}{4}$  inches in circumference. The aortic valves become finer, form a sort of fibro-cartilaginous fold, rigid and  $3\frac{1}{2}$  to 4 lines in height, (in place of about  $7\frac{1}{2}$ .)

The cavity of the left ventricle is smaller than that of the right, though rather larger than normal. Its walls towards their base are about 7 lines thick. The left ventricle is dilated and thickened; its internal membrane is opaline and composed of several layers.

The auriculo-ventricular orifice is so contracted as hardly to admit the point of the ring finger. The two plates of the valve are united so as to form a ring, and are transformed into a fibro-cartilaginous tissue. On both sides the surface is roughened by calcareous concretions and horny productions,

some of which protrude from  $2\frac{1}{2}$  to 3 lines, and are suspended like stalactites. On the side of the auricle the right leaf of the valve is transformed almost entirely into a calcareous concretion, friable and very hard to the touch. The valvular tendons are thickened, shortened, and the summits of their fleshy columns are themselves transformed into a tendinous tissue.

The pectoral aorta is perfectly healthy.

Is not this autopsy, in some sort, an anatomical translation or paraphrase of the diagnosis? Pericardial adhesences had been suspected, and they were verified; bicuspid contraction and aortic insufficiency were announced, and both existed. I may perhaps fall into useless repetitions, but I cannot resist the desire of drawing the parallel between the physical signs and the basis.

Souffle of the first time; contraction of the aortic orifice, and bicuspid insufficiency.

Souffle of the second time; bicuspid contraction and aortic insufficiency.

In reflecting on the contraction of the two orifices, we explain without trouble, the tone of the souffles, that double saw sound, that puff of steam, that aspirant sound of the second time in the aortic region.

In reflecting on the cretaceous state of the bicuspid valve we comprehend that raspy sound, so often verified.

In the aortic region, the souffle of the first time declined, because we had then only the souffle produced by the aortic contraction, that which the bicuspid insufficiency produced being, doubtless, too feeble to reach our ear; the second, on the contrary, predominated because it was the product of both the contraction and the cretaceous state of the bicuspid, of which the sound was able to reach the ear, and of aortic insufficiency. This second souffle was strong enough to mask all clacking and did not permit us to hear that of the pulmonary valves; the first souffle, more feeble, allowed us, on the contrary, to distinguish, but distant and obscurely, the clacking of the tricuspid orifice, of which the dilated valve was not insufficient.

Finally from our anterior observations, save the last, the infiltration that we had noted during life gave us reason to expect a notable dilatation of the right auricle and this lesion was not in default.

OBSERVATION 19.—*Clots in the heart. Considerable dilata-*



*tion of the right heart. Aortic vegetations. Bicuspid contractions.*

Bouillon, 41 years old, brewer's servant, was received on the 2d of March, 1838.

He is of a strong constitution, heretofore in good health, and declares that he has never had any other maladies than three attacks of rheumatism, of which the last, and the gravest, occurred eighteen months since.

Eleven months ago he experienced dyspnœa and palpitations for the first time, Nine months since a little swelling of the feet occurred, now disappearing and anon recurring, until during the last two months it has become permanent. For some days past he has been a prey to a very pronounced dyspnœa. He has had many attacks of faintings with complete loss of consciousness.

*Present condition.* The patient is seated on the edge of his bed, his legs pendent and supported on a chair. The respiration is panting 40 to 44. The face is pale and little livid; the lips large and bluish; the eyes prominent; the jugulars distended, especially in expiration; the inferior extremities strongly infiltrated and rather cold.

The precordial region, more prominent than the opposite one, is overrun by some venous branches which are not observed on the latter. The beatings of the heart are much more extended than natural, but embarrassed as if the organ was confined to a narrow compass. The point of the heart is felt  $2\frac{3}{8}$  to  $2\frac{3}{4}$  inches below the nipple, and quite without, so as to be transverse. The dullness is  $4\frac{3}{4}$  inches vertically and about  $6\frac{1}{4}$  transversely. The hand feels a slight tremor.

In the region of the left cavities a double rasp or saw sound replaces, perfectly, the valvular tic-tac, and extends into all the precordial region and the environs. The soufflé is stronger and more prolonged in the first time. The pulse is small, very difficult to feel on the left side, more distant on the right, irregular, unequal and intermittent. We count from 124 to 128 pulsations, of which some escape by their smallness; others are a little vibrating. The pulsations of the heart seem to be less unequal and irregular than those of the pulses.

In view of all these morbid phenomena, M. Bouillaud diagnoses; *Enormous hypertrophy of the heart. Thickening, induration and insufficiency especially of the left valves.—Clots in the heart.*

After the detailed diagnosis of our preceding observations

the reader may, perhaps, be surprised at not finding the same precision in the terms of this. The last element of this diagnosis is, nevertheless, of such a nature as to explain fully this absence of localization to all who are in the habit of ausculting the heart. And, in fact, when the blood concretes in the interior of its cavities, there often results no less disorder in the sounds of the organ, whether normal or morbid, than derangement of its functions.

Now, since it is the first time that the occasion has offered us an opportunity to particularize the symptoms of these sanguineous concretions, let us say, in a few words, what determined M. Bouillaud to admit them here.

Let us remark, first, that this patient presents to us the most intense orthopnœa that we may have yet encountered. This anhelation, this anxious countenance, this distension of the jugulars; all these signs, in fine, of a considerable restraint in the venous circulation and especially the long duration of all these morbid phenomena, the prolongation of this stasis of the venous blood, is there not already great reason to suppose, the blood being checked in its course, that the coagulation must be an imperious necessity? But let us come to some more formal indication. These indications are furnished to us by the restraint in the movements of the heart, movements extended, but which, according to M. Bouillaud, seem yet in a narrow compass; the inequalities and irregularity of these pulsations; and finally the characters of the pulse, generally small, sometimes imperceptible, at others, on the contrary, almost vibrating, more unequal and irregular than the heart itself. Then must there be, between the radial pulsation and the impulsion of the left ventricle, independently of the organic lesion of the valves themselves, some other obstacle, sometimes fixed, but often moveable, breaking and intercepting more or less completely the effort of the sanguineous wave.

Such are, in fact, the principal signs of clots in the heart; signs very important to be known, for they may give us a key to functional disorders, which without them we would not know how to explain; and at the same time, moreover, suggest to us an active and energetic medication, as powerful against these temporary disorders as powerless against the organic lesions properly so called.

Let us now return to our patient.

The following days a little amelioration declared itself under the influence of a bleeding and digitalis, but the infiltration made new progress; the scrotum and the penis became

œdematous; the cavity of the peritoneum filled with serum; the pulse remained small and even more and more imperceptible; the sounds of the heart, always accompanied by a double souffle, became heavier and more smothered; the dyspnoea reappeared; so violent and frightful, that the patient, almost erect on his bed, demanded to be strangled rather than be left to suffer thus; finally, on the 27th of March, death came to put an end to this frightful agony.

*Autopsy.* The pericardium is uncovered to an extent of 7 inches transversely. It contains about the fourth of a glass of turbid serosity. The heart, of which the point is turned almost directly to the left, is distended so much as to present the appearance of a calf's heart. These clots are, in part, soft and discolored—half organized. Freed from these concretions, the heart, with the origins of the large vessels, weighs nearly 25 ounces.

The right ventricle is dilated so as to be capable of containing the fist. It is hypertrophied in proportion to its dilatation. The parietes are, in their maximum thickness, about 5 lines.

The right auricle is dilated and thickened nearly in the same proportion. The tricuspid orifice has a circumference of nearly  $6\frac{1}{2}$  inches. The valve moreover presents its normal conformation. It is thickened, hypertrophied, nearly sufficient.

The valves of the pulmonary artery are thin, larger than natural. The circumference of the orifice is over 4 inches.

The left ventricle is dilated so as to contain a duck egg. Its parietes are about 9 lines thick, and, what is very remarkable, they are much less firm and of a less lively red than the parietes of the right ventricle.

Water poured into the aorta approximates the valves and does not penetrate into the ventricle. These valves are quite well shaped and thickened; their free border is strewed with small granular vegetations, looking to the ventricular cavity, organized under the fever of venereal warts. The circumference of this orifice is  $3\frac{1}{2}$  inches. The mitral valve offers a very considerable thickness and is transformed into a fibro-cartilaginous tissue. The orifice that circumscribes this valve is contracted so as to admit the extremities of two fingers. Its circumference is  $3\frac{1}{2}$  inches. On the auricular side the thickened valve forces a sort of wrinkled fold, like the skin, about the anus. The borders, where the tendons are inserted, present small vegetations, but less numerous.

The parietes of the left auricle are thickened in the same proportion as the corresponding ventricle; its internal membrane offers rather an areolar aspect, and is a little thickened.

The reflections we have heretofore made will free us from the necessity of comment on this autopsy. We comprehend, without trouble, that without the clots of which we have specified the effect, it would not have been less possible here than in our previous observations to perceive that narrowness, so notable, of the bicuspid orifice, and these vegetations of the aortic valves; the cause of the vibratory tremor, and their insufficiency, and perhaps even that insufficiency and that enormous dilatation of the tricuspid orifice and of the corresponding auricle, coinciding again here with a considerable infiltration and with œdema of the lungs, that I also see noted in the autopsy of which I have thought it my duty to reproduce only so much as related to the heart.

We perceive in this fact an example of fixed sanguineous concretions, if I may be allowed thus to speak, persisting and surviving the subject. In the following observations we will encounter those of a temporary character. This is one of the reasons why I think it proper to add it to the preceding cases.

*OBSERVATION 20.—Temporary Clots. Contraction and a little bicuspid insufficiency. Slight aortic thickening.*

Frederic Piquois, founder, aged 36, entered our service 23d December, 1841.

This patient, of quite a strong constitution and lymphatic temperament, says he has habitually enjoyed good health. He is only subject, since eight or ten years, to headache and giddiness. During the past year there has been added a cough, suffocation in ascending, œdema, which at first limited itself to the left jaw, and attacked afterwards the superior extremities, then the inferior of the same side, and since six weeks the right inferior extremity, and finally vomiting which has become more frequent during the last few weeks. All these symptoms have obliged him to cease from his occupation for six months, and even several times to keep his bed.

*Present condition.* The face, the trunk itself, the fore-arm, the left wrist, and the two inferior extremities are swollen and doughy to the touch. The inferior extremities, in particular, preserve deeply the impress of the finger. The skin is moreover of a dull tint generally. Some nausea at intervals. The epigastrium and abdomen indolent. No pain in the renal region. Urine scarce. The penis a little œdematous. Heat of the skin little raised. The pulse rapid, unequal, scarcely



seizable at 120 to 124, contrasting by its smallness with the force of the subject. The precordial region presents a prominence of about four degrees, but the pectoral parietes, of the left side especially, are a little infiltrated. In consequence of this circumstance, without doubt, it is difficult to find the point of the heart, and its beatings are irregular. We nevertheless finally succeeded in perceiving them in the sixth intercostal space, from 10 to 15 lines without the nipple. The pulsations of the heart are scarcely felt by the hand firmly pressed over it, and there is no appearance of vibratory tremor. The dullness has a vertical extent of about three inches and  $3\frac{1}{2}$  transversely. In ausculting the sounds of the heart we remark that the first seems to be effaced, whilst the second is drier than natural. In the origin of the aortic orifice and the aorta this contrast is at its maximum, the second sound being then especially hard and like parchment. In the region of the auriculo-ventricular orifice the same absence of the first clacking, the clearness of the second being less sensible, and besides, especially at intervals, a little souffle, quite rude, mingling itself sometimes with the first, at others, with the second sounds. This souffle is not propagated into the carotids, nor is there any *visible* distension of the jugulars. The respiration is embarrassed and with difficulty limited to 28 per minute. There is only a slight sub-crepitant rale in the inferior and posterior part of the left side.

The next day M. Bouillaud verified the following :

The pulse is at 124, felt with great difficulty, but is tense and without notable irregularity at this moment. As yesterday, the point of the heart is felt quite obscurely in the sixth intercostal space, a little without the nipple, and the first clacking is superceded by a souffle. The second is rude, of a slight parchment tone, and at present without souffle. The souffle disappears as we recede from the left auriculo-ventricular orifice, and, towards the clavicle, is heard a single sound corresponding to the second time.

M. Bouillaud dictated then the following diagnosis :

*Very considerable hypertrophy of the heart (about 14 pounds). Induration and thickening of the left valves, with deformation of the bicuspid and contraction of the corresponding orifice.*

Evidently, in making the diagnosis, M. Bouillaud takes into consideration both that which I had verified the day before and what he himself observed. The first clacking was superceded by a souffle, growing feeble as we receded from the bicuspid



orifice; then there was deformation of the bicuspid valve, and even a little insufficiency. In this same region there existed no souffle at the moment, but I had verified it the evening before; then there might be a certain degree of contraction of that orifice, a lesion which coexists so often with the deformation and insufficiency of a valve.

Is this diagnostic definition, and does it give the key to all the symptoms observed? No, doubtless.

The dyspnœa, the general infiltration, the irregularity of the pulsations, observed at least at intervals, the smallness and especially the rapidity of the pulse (124) indicated certainly a notable obstacle to the venous circulation. But what was that obstacle? Perhaps a right ventricular lesion, but possibly also some sanguineous concretions. Certain ulterior signs ought to decide this question. Let us remark, moreover, that this anasarca of our patient had not followed its usual march. It appeared to have begun at the face and reached the inferior extremities only in the later periods. Was this not a reason for making the inquiry, whether there had not existed, as a complication of the disease of the heart, and as an essential cause of the serous infiltration of the extremities and trunk, that affection of the kidneys, called the *disease of Bright*? Under this impression M. Bouillaud directed the patient to preserve his urine.

The next day it was found pale, opaque, slightly acid, a little frothy, nitric acid and heat producing a white precipitate nearly of the consistency of the syrup *orgeat*. The patient had vomited several times.

We add to the diagnostic: *albuminuria*.

These morbid phenomena, and especially the vomitings, persisted almost constantly until the death of the patient, which event they doubtless hastened. But the sounds of the heart presented an important modification, which should be noted.

On the 30th and especially on the 31st of December, and consequently a few days after the entrance, M. Bouillaud, in ausculting the precordial region, was much astonished to find the two sounds of the heart quite distinct, especially the second, always resounding and of a slight parchment tone, the second being only a little rude; further, there was none, or only a slight and soft souffle in the first time, particularly in the aortic region. The apex of the heart is thrown into the fifth intercostal space. There is no oppression in a state of repose. The swelling of the extremities has diminished.

Finally let us add, for this is essential, that the pulse has fallen to 60.

I repeat, that this last fact is of the highest importance; for it no more permits us to doubt that at the entrance of the patient, the pathological phenomena, that we have indicated above, were caused by clots of blood which produced, among others, this triple effect:

1st. To distend the heart sufficiently to cause its point to descend to the sixth intercostal space.

2d. To restrain the valvular play, so as to suppress the clacking of the bicuspid valve, much more submitted to their contact than the aortic valves, in consequence of its conformation and of its more central position.

3d. To produce on the part of the ventricular contractions and consequently in the sanguineous wave itself, an exaggerated quickness, a condition so favorable, perhaps almost essential here, to the excess of friction (*frottement*) which all species of *bruit de souffle* presuppose.

M. Bouillaud now completes the diagnosis by this last feature.

*A probability of temporary polypi-like concretions in the cavities of the heart.*

But we may be asked, perhaps, if these clots are once admitted as the cause of the morbid phenomena noted during the first days, and which have, in fact, disappeared since, what will become of our first diagnosis relative to the bicuspid and aortic valves?

I respond, we will still maintain that diagnostic. And why? Because if it be true that the *souffles* have disappeared, save that of the first time, which rigorously speaking may, perhaps be considered a *souffle* of anemia, it is not less true that the valvular sounds have not rebecome normal.

The first sound is yet a little rude and the second has a parchment tone; then the corresponding valves are not sound. Do we desire yet new proof of this, which will triumphantly demonstrate the validity of our preceding considerations? I find it in the course of this observation.

February 14th. The swelling has made new progress; the pulse has attained 92; and we find a very distinct raspy *souffle* in the first time.

February 23d. There is considerable infiltration, even ascites for some time past; pulse 88; the raspy *souffle* persists.

Further, in order to arrive, finally, at a still more positive

demonstration, let us now seek, in the necroscopic lesions the proof of our diagnostic.

The preceding reflections will allow me to content myself with their exposition, without comment. It will be readily perceived, that our diagnosis was correct in all points.

Our patient died on the 4th of March.

*Autopsy.* Considerable infiltration of the extremities and trunk.

*Thoracic cavities.* Very notable serous engorgement of both lungs and especially of the left.

The heart is hypertrophied, but to a medium extent. It weighs, with the origins of the large vessels, nearly 17 ounces; the hypertrophy particularly involves the left ventricle.

Neither the pulmonary or tricuspid orifices, nor the right ventricle offer any thing special.

The left ventricle is enlarged; and its parietes present, from the base almost to the apex, a thickness of 9 lines. Its columnæ-corneæ, sufficiently robust, are inserted, by rather short tendons, into the bicuspid valve, which is sensibly puffed at its free border, and which appears a little shrunken and narrower than natural. In fact the circumference of the corresponding orifice is only  $3\frac{3}{4}$  inches (instead of  $4\frac{1}{4}$ ).

The circumference of the aortic orifice is  $3\frac{1}{2}$  inches; its valves are quite well-formed and sufficient, (which is proven by pouring water into the vessel.) They present only a sort of small fibrous patch towards their base; and, above their free border the internal membrane of the aorta presents a line, almost circular, of a nankin yellow, which divides, more by its color than by its feeble relief, the smooth and polished surface of the arterial tunic.

*Abdominal cavities.* Both kidneys are remarkably small; they resemble those of an infant. Their vertical diameter is 4 inches, and the semi-circumference, taken from the middle of their convex edge to the center of the fissure, is  $2\frac{1}{2}$  inches. They are equal in weight. United together with their fibrous capsule and a portion of the ureters 4 or 5 inches long, they weigh about half a pound. They present nothing remarkable interiorly, but on their exterior they are granular, red, and in many points adherent to their capsules which cannot be removed without rupturing. The alteration indicated, occupies in some manner, only the superficies of the

organs, which at the depth of half a line resumes its normal texture.

The ureters and bladder are remarkably pale. The urine contained in the bladder was collected with care and treated with nitric acid, and there resulted a very abundant white precipitate.

Let us now finish this analysis, perhaps already too far extended, by a last observation interesting, less on account of the anatomical lesions, the same as those we have already seen very often, than even the existence of a symptom that we have not yet encountered, the triple and quadruple sound. We see, moreover, death produced by a clot in the heart. This is a new feature in the history of that morbid product; and also a point of contact between it and the cases we are about to relate.

This observation is one of those, which I collected under the dictation of M. Bouillaud before I entered his service.

**OBSERVATION 21.**—*Contracted, insufficient and calcareous condition of the bicuspid valve. Hypertrophy of the left auricle. Triple sound. Death by a clot.*

Garret, a butler, aged 38, was received into our service on the 4th of January, 1838.

He has rather a delicate constitution, and a lymphatico-nervous temperament. Heretofore, usually in good health, he has complained of palpitations only since about one year; and no antecedents appear to account sufficiently for the invasion of an organic disease of the heart. The articulation of the left shoulder is in a semi-anchylosed condition, with a cicatrix towards the middle of the arm, which seems to have been the consequence of a violent phlegmonous inflammation, that occurred at the age of 12 or 13 years. The entire limb is a little atrophied. The patient himself attributes the palpitations of his heart to an influence which lasted five weeks.

*Present condition.* (Noted by M. Bouillaud). The face exhibits lassitude and is pale as well as all the remainder of the skin. There exists neither œdema of the extremities, ascites, or dilation of the jugulars. The pulse is rather small, but distinct and regular, at 56–60. No very distinct prominence. The apex of the heart strikes the sixth intercostal space and forcibly repels the finger. In all the precordial region, properly speaking, that is to say, from the base of the heart to its apex, we perceive great vibratory tremor. The corresponding dulness is  $4\frac{1}{2}$  inches vertically and 5 transversely. The valvular sounds are replaced by a double souffle;

the second more prolonged than the first, is accompanied by a sort of scraping, or snoring, similar to that attendant on sleep. In ascending along the track of the aorta this double souffle is heard but without mingling with the snoring alluded to. Further, there is about the heart an amphoric tinkling which is heard at a distance, and is analogous to the sound of boiling water. Receding from the precordial region, the double bruit de souffle ceases to be heard and the amphoric sound disappears. The double souffle propagates itself to the posterior part of the left side of the chest. There is no vibratory tremor in the arteries. Respiration is perfect.

M. Bouillaud dictated the following diagnostic:

*General hypertrophy of the heart (about one pound). Thickening and induration of the left valves, with a contraction of the auriculo-ventricular orifice.*

The next day a very distinct triple sound is heard in the precordial region which may be traced as far as to the right side of the chest. This is the result of a decomposition of the second sound, and is accompanied by the souffle already noted.

The amphoric sound of the previous evening is no longer distinguished. The pulse is only 48-52; it is always tense and vibrating and not increased.

During the following days, there is persistence of the triple sound which is even more and more distinct. The two last sounds do not occupy more time than the first, which is synchronous with the pulse. The last is always made up by a souffle. In the track of the aorta there exists only the usual double sound.

January 23d. The triple sound extends into the carotid. Under the influence of digitalis the pulse has fallen to 32. During the examination the patient was restless, and, a few moments after, M. Bouillaud discovered four sounds, which were soon superseded by the usual triple sound.

The 31st. The triple sound persists ever with a raspy souffle which ends it. It may be represented by tic---tac---s-s-s-s. M. Bouillaud made some remarks on this subject, and explained the third sound by saying that, without doubt, the left auricle emptied itself through a contracted orifice, when the ventricular diastole was already accomplished. This morning, the pulse has risen to 48. The patient is affected by very intense salivation which he has contracted by mercurial frictions on the pubis. February 4th. He died at the



moment of our visit; no signs had foreshadowed this sudden death.

Previous to developing the results of the autopsy, let us for a moment, review some of the peculiarities of the symptomatological history of this patient.

Is it necessary to state the grounds for the diagnosis? With this double souffle, so rude, that it is accompanied by a vibratory tremor evidently located in the region of the heart itself, so strong, that it propagates itself as far as to the posterior part of the chest, and that the valvular clacking can nowhere be heard; who does not perceive, as a consequence, that both orifices are affected, or, if only one, that it is injured in a very high degree? If now we consider that the rudest of these souffles, which is mingled with a sort of scraping, does not offer this accompaniment along the track of the aorta, ought we not to conclude that this bicuspid orifice is the point of departure of this morbid sound? Finally, since this sound is heard in the second time, should we not conclude also that the lesion of this orifice is especially a contraction?

In reference to the amphoric tinkling noted only on the first day, had it any relationship with the lesion which we are about to diagnosticate? Was it, for example, a phenomenon of the same nature as the vibratory tremor? I do not think so. I prefer to view it as a peculiar sound, of which I will speak with some minuteness hereafter, which, although studied in other regions, has not, to our knowledge, been described as existing in the precordial. I allude to the *bruit rotatoire*.\* I am justified in this idea by the description dictated to us by M. Bouillaud, without pronouncing on its cause, and especially by its feeble propagation beyond the precordial region, and finally, its prompt disappearance.

One word now on the triple sound of which this observation offers us the first example. We will speak hereafter, of the different interpretations, of which this curious modification in the rhythm of the sound of the heart, is susceptible. Let us remark here that on which M. Bouillaud dwelt, and which we will presently see fully sustained by the autopsy; and let us note this peculiarity that, for the triple sound to be heard, the succession of the sounds of the heart must probably present a certain degree of slowness, since it did not exist on

\* A sound attendant on the first sound of the heart, and so called because it was thought to resemble the rumbling of distant wheels. It has also been attributed to muscular contraction, and is hence sometimes termed *bruit musculaire*.—*Translators*.

the first day when the pulse was 56-60, and became perceptible the next day, the pulse being at 48, and that afterwards it appeared to become more and more distinct as the pulse gradually diminished in frequency. May this not explain why we have not before encountered this triple sound in cases so anatomically analogous to the present? For if I am not deceived, no case has presented a similar slowness of pulse.

Finally, let us come to the recapitulation of the cadaveric lesions.

*Autopsy*, (25 hours after death.) The pericardium is exposed to the extent of about 5 inches square.

The heart, lying almost transversely, is nearly triple its normal volume, depending in part on the distension of its cavities, especially of the auricles, by sanguineous concretions. In fact, on dividing the vessels of the base of the heart, an enormous quantity of half coagulated blood was discharged from the auricle, and this evacuation reduced the volume of the heart at least one-third.

The right auricle still retained an unctious looking clot, of a yellow amber color, of the size of an egg, prolonging itself into the right ventricle, where it is one-half less. M. Bouillaud believes this clot to have been the immediate cause of death. Freed from all its concretions, the heart weighed, with the origins of the large vessels, about 14 ounces.

*Right heart.* The valves of the pulmonary artery are thin, large and well formed. The circumference of this orifice is 4 inches. The cavity of the right ventricle is dilated to a medium extent; its parietes are less than 5 lines in thickness.

The right auricle is dilated, and might contain a large egg; but its parietes are of a good thickness at every point, its columnæ-corneæ well developed and hypertrophied. The tricuspid orifice has a circumference of 6 inches. The valve preserves its normal shape; it is evidently enlarged and a little opaque.

*Left Heart.* The aortic valves are sufficient, well formed, a little hypertrophied, especially at the middle of their free border, and of a tint rather opaque and lactescent. The circumference of the aortic orifice is nearly 4 inches.

The cavity of the ventricle is but slightly or not at all dilated.

The bicuspid orifice is transformed into an oval aperture, capable of admitting the end of the little finger, and of which the large diameter is nearly 10 lines, and that of the small

rather more than 5. The two laminae of the valve, adhere by their angles, and circumscribe the orifice. They are thick and fibro-cartilaginous. At the posterior part, they are more than 5 lines thick, and in consequence of the presence of a calcareous concretion, as seen from the auricle, it is divided into two valves so as somewhat to resemble a shell. The edges are rugose and unequal, made up of calcareous granulations. The columnæ-corneæ, which are inserted into the valve, are strong and their tendons hypertrophied. The summit of these columns are transformed into tendinous substance. The endocardium adjacent to the valve is thickened, and changed into a fibrous tissue. It is unequal and resembles shagreen. Separated at its circumference, the valve weighs 180 grains.

The auricle is so much dilated as to be capable of containing a goose egg. Its parietes are generally thickened ( $1\frac{1}{2}$  lines) the result of the hypertrophy of the muscular tunic, which is of a deep red. The endocardium is easily detached, and is nearly double its normal thickness.

The origin of the aorta is slightly dilated, and sown with spots of yellowish white, without calcareous incrustations. At certain points we remark a pale red color. This same color is also observed, and it is even more marked on the aortic valves.

There exists no traces of cadaveric imbibition.

This autopsy agrees so well with its semeiology and with the diagnosis, that it cannot suggest any very numerous reflections.

There is perhaps but a single point which demands our consideration: viz: the very considerable dilatation of the right auricle, coincident here with the complete absence of any notable infiltration. Is this fact calculated to invalidate our antecedent observations? I think not; and why? Because here we have not only a dilatation of this auricle, but also a proportionable hypertrophy of the muscular tissue; the tricuspid orifice is adequately enlarged and closed by a well formed valve; finally, the right ventricle is not dilated, and the pulmonary orifice itself is quite free; all, circumstances but little favorable to that stasis of the venous blood which is the cause of infiltration. In our antecedent observations we had what may be termed a passive dilatation; here, we have in some manner a veritable active aneurism, probably the result of that inflammation of which we have found so many scattered vestiges, in the thickening of the endocardium, the

redness of the aorta and in the amber colored and fibrous concretions, the formation of which must have been promoted by the stomatitis of our patient; aided, moreover, as it undoubtedly was; by that obstacle, so evident, to the arterial circulation, located at the bicuspid orifice, the approximate, if not the only cause of the dilatation of the left auricle, and the influence of which was gradually extended to the right heart, the obstructions in which, as we have seen, appear to have produced the asphyxia of the patient.

Here ends the first part of this memoir; the exposition of facts. I might readily add to this list, many facts not less authentic than the preceding. But I am fearful of fatiguing my reader and of falling into wearisome repetitions. I think I have exhibited almost complete, all the chief alterations, or at least the most common that the heart can present, and afterwards sufficiently demonstrated, by cadaveric examinations, the possibility in all cases, of a positive and exact diagnosis. Have we not seen, in almost all, as perfect agreement between the diagnosis and the autopsy, and, when there has been an omission, or even an error on our part, have we not frankly confessed it, in such a manner as to promote the interest of the science—an error clearly pointed out, being the best precept for avoiding future mistakes of a similar character?

Let us now recur to all these facts in order to unfold and to study successively the means of explanation, by the aid of which we have succeeded in analyzing, so minutely, diseases heretofore reputed as impenetrable. But, before we approach this didactic part of our subject, let us take a general glance at the divers lesions which we have enumerated, and see if this view of the ensemble will not give rise to some considerations which have not yet occurred to us.

And, first, let us observe the wonderful difference in the frequency, and especially in the gravity of the affections of the pericardiums and those of the heart itself. Of our twenty cases, there is not an example of open acute pericarditis, or of considerable effusion in the envelope of the heart, capable, of itself, of producing a fatal termination. I have followed, for nearly six years, the clinic of M. Bouillaud, and during all that time I have observed, only five or six times, the complication here alluded to. I have not cited these facts because my object here was to study the heart itself, rather than its serous envelope, of which the symptomatological history is well known, and because, under the aspect of the subject which occupied our



attention, these facts could not have added any thing essential to our observations.

If, as I have just remarked, the grave lesions of the pericardium are very rare, the same is not true of latent lesions which are revealed to us at the autopsy in the form of slight and circumscribed adhesions or small, prominent and isolated plates. These, if I may be allowed the expression, are almost necessary accessories of grave lesions of the heart, and which, in our necropsies, have rarely been in default, although we may have generally passed them in silence, except when these adhesions, or false membranes were of such a character, as to become themselves causes of functional disturbances, or give rise to a suspicion of their existence by certain palpable signs, as in our observations 9, 13, 14 and 18.

Let us now come to the heart, properly speaking. The first remark by which we must be struck, is the singular predominance of the left over the right heart, in respect to its morbid alterations.

True, we have often seen the right heart participate in the hypertrophy of the left; but has there not always been a difference between the two, sufficiently marked to render it apparent that the hypertrophy of the left heart had been the capital and primitive lesion, and that of the right only accessory and secondary? Have we not encountered valvular alterations almost exclusively in the left heart, those in the right consisting, most frequently, only in a passive enlargement of the orifices, especially that of the tricuspid?

What can be the cause of a difference, so decided, between two cavities bordering on each other, and which are so analogous in their structure? It can be owing only to the difference in the qualities of the blood which traverses them respectively. Approximating each other anatomically, the right and left cavities are very far removed from each other in physiological point of view.

They differ as widely, in this respect, as arterial and venous blood. Relative to the pathogeny of the affections, which engage our attention, I rank myself, without hesitation, by the side of M. Bouillaud; the inflammation of the internal membrane of the heart, the sanguineous congestion of the different parts of this organ, and I will add the molecular composition of the blood which flows through it, appear to me to be the essential points of departure of the structural alterations which the muscular tissue and the valves present to us.

There is, nevertheless, still another circumstance, which,



should enter into the account, in studying the anatomo-pathology of the centre of circulation. This circumstance, secondary, it is true, and which always presupposes some other previous morbid condition, is the force of the blood in the interior and on the walls of the cavities of the heart—an effort of dilation altogether mechanical, and from which results the enlargement of its cavities and their orifices, but which, “it is well understood, does not exclude, for the explication of what has been termed eccentric hypertrophy,” an exaggerated nutrition submitted fortuitously, perhaps, in the pathological growth of the organ, to the same laws as in its normal evolution, that is to say, having simultaneously augmented its substance and enlarged its capacity.

This mechanical dilation occurs, especially, in those partial enlargements which either of the cavities or orifices may present to us, beyond which is the free flow of the sanguineous wave finds itself more or less completely intercepted. I have said, that the right heart offers those passive alterations, if I may be permitted thus to designate them, more frequently than the left. One word now on the mechanism of their formation.

If it were common to encounter an organic lesion of the pulmonary artery, and a contraction of the corresponding orifice, we should readily conceive that, behind this obstacle, the right ventricle and the auricle itself must become dilated. But as we may determine for ourselves, by the facts that we have passed in review, this lesion of the pulmonary valves is of very rare occurrence. And yet we have very often witnessed a dilation of the right cavities. Why? because, according to M. Bouillaud, an obstacle, situated in the left cavities, gradually exerts on the circulation of the right, an influence which, though distant, is not less sure and real. Let us developpe this idea by giving it the support of an example.

A lesion exists at the orifice of the aorta. This lesion arrests or at least, impedes the passage of the blood projected by the left ventricle. A portion of this blood remains then in the ventricle and becomes an obstacle to the ingress of that which should be received from the auricle of the same side. This auricle in its turn retaining a part of the blood in reserve, the pulmonary veins, which there terminate, are, for the same reason, only imperfectly emptied; as a consequence, the same must be true of the pulmonary parenchyma which sustains them. If this parenchyma remains thus charged with a quantity of blood, which should have passed freely,

forward, it cannot receive all the venous blood, which the ramifications of the pulmonary artery conveys to it, and hence, retrograding in its turn, the venous wave must distend, successively, the pulmonary artery, the right ventricle, the auricle of the same side, and eventually the entire venous system, from which soon result serous infiltrations, sometimes partial, at others general.

Let us remember, that by duly considering the extent or the importance of the serous effusions, that we have, more than once, diagnosticated dilations of the right heart, and especially of the auricle, diagnosis afterwards verified by the autopsy. Let us add, that if at the beginning of this arrestation of the venous circulation, the right ventricle can redouble its efforts, and, by this excess of the muscular contraction, cause the palpitations and dyspnœa of which the afflicted complain at this period; if it contributes then without doubt to hypertrophy its tissue, a moment may, nevertheless, arrive, when its dilation will be such, that having lost in contractile force what it may have gained in capacity, the feebleness and impotency of its contractions will become a new cause of stasis of the blood, added to those we have already pointed out, and strengthened, moreover, by another morbid circumstance, of which I have not yet said any thing, and which is the result of the dilation of the right cavities: I allude to insufficiency of the tricuspid valve, which we have proven many times after death, and even announced its existence during life.

Thus, then, to sum up what concerns the right heart according to our observations: most frequently, I will not say constantly, eccentric hypertrophy, passive dilation, and often simple hypertrophy, that is to say, without any very notable valvular lesions, constitute its anatomo-pathological history.

In the left heart, on the contrary, hypertrophy, without valvular lesions, is almost an anomaly. We have cited one example of it, (Observ. 1st,) and yet in that case we could only perceive that the valves were not perfectly round. As in the right heart, so here, the auricular orifice is most frequently injured or the lesion predominates at this orifice when there are coincident aortic lesions. More than once we have shown the probable reason of this predominance in the influence, which the *chordæ tendinæ*, that are inserted in the bicuspid valve, may exert on the coagulation and deposition of the fibrin of the arterial blood. As in the

right heart, we often meet with partial dilations, sometimes of the ventricle, at others of the auricle; but we have less frequently found the orifices free or dilated in the left than in the right heart. The contraction of these orifices is certainly one of the most common lesions which we have observed, whether there existed concomitant insufficiency or not. As, apropos, may I not refer to that peculiarity, curious, perhaps, to those who believe in final causes, that by the arrangement even of the morbid lesions which the two hearts present to us, and of which we have spoken, it seems that nature may have, in some manner, endowed herself, by a great foresight, so that the fatal term, inevitable sooner or later as a consequence of the disease, might be, at least as much as possible, procrastinated? I will explain myself.

Of all the mortal perils which may result from hypertrophy of the heart and lesions of the valves, there are two principal ones—an afflux of too large a quantity of blood to the brain, producing apoplexy, and too great a diminution of the quantity sent to the lungs, causing asphyxia. I repeat that such as they usually present themselves, the lesions of the orifices seem to be, at least, as favorable as possible to the prevention of this double accident.

Thus the right orifices, which lead to the lungs, are, as we have seen, almost always free and open. Thus, also, are not the left orifices which open towards the brain, most generally contracted or insufficient; so that, as a consequence, there is an imperfect issue of the sanguineous column at the first time, through the contracted aortic orifice; or a division of that column itself by a reflux movement, sometimes into the auricle in the first time, and at others into the ventricle in the second time.

I repeat it, this peculiarity is, in my opinion, a curious and fortuitous occurrence, rather than a precaution of nature, a vestige of order in the midst of disorder; but I dwell upon the fact, in passing from this motive only—that the fact should not be entirely neglected, in relation to prognosis, and in a general study of the diseases of the heart.

Let us now begin the examination of our means of exploration, not for the purpose of composing here a complete treatise on diagnosis, but especially to indicate the practical views which the clinical facts themselves, that we have analyzed, may suggest.

## SECOND PART:

### *A succinct consideration of the means of Diagnosis in diseases of the Heart.*

The first sign which we have remarked almost constantly in advance of those of which the ensemble constituted our symptomatological history, was the state of the pulse. There is, as we know, two things to study in the pulse; quantity and quality. It is generally essential to note the quantity only in the acute affections, pericarditis, endocarditis, &c. In those cases the pulse may attain an extreme quickness—it has surpassed 130 and 140. The twenty cases related above have not furnished one of this kind.

I will refer only to the fact of sanguineous concretions (Obs. 19) in which the heart beat 124 times per minute, as if to compensate, by the multiplicity of its contractions, for the insufficiency of each of them. In those cases then, there was generally nothing remarkable about the pulse in this first point of view. But this is not the case in reference to its qualities.

The qualities of the pulse should, doubtless, be properly estimated in the acute disease, and most especially in the chronic affections of the center of the circulation. Let us remember that more than once, the pulse has been the first index we have had of an affection of the heart, and has awakened within us a suspicion, transformed by the subsequent examination, into a certainty. We have sometimes seen a vibrating, developed, and resisting pulse immediately announce to us (see Obs. 5 and others) the existence of an hypertrophy of the heart and particularly of the left ventricle; and from this development of the pulse we have been led to conclude that there was apparently neither pronounced contraction of the orifices, nor very considerable valvular insufficiency, and far less a great hypertrophy of the arterial system. Sometimes the opposite characters have caused us to foretell oppo-

site lesions. Almost always we have been able to discern a perfect accordance between the state of the pulse and that of the heart; it seemed that the first was but the echo of the second. We have, however, sometimes noticed between the two signs a discordance which became of itself a new sign, announcing, for instance, the presence of some obstacle between the ventricular contraction and the radial pulsation. (Obs. 19.)

Have the qualities of the pulse been modified in all of our cases? No, unquestionably not: and it is well to be aware of this practical truth, that, if certain disorders of the pulse correspond often to certain lesions of the heart, it is no less true that the absence of these first does not necessarily suppose the absence of the second. With some of our patients the pulse remained regular up to the last moment. This brings us to the reflection, that if the ancients—deprived, it is true, of such positive physical signs as more modern times and other methods of examination have furnished us—considered the pulse as truly the compass of the observer in the study which now occupies us; that if Corvisart himself thought proper to develop with so much pains and so minutely all the indications of the pulse, we must acknowledge that the progress of science has not sanctioned, in every respect, their confidence and their praise. Who is there at the present time would dare to diagnose a lesion of the heart, on account only of the intermissions or irregularities of the pulse? Deceptive characters which may coincide with lesions purely dynamic or nervous.

I know that it has, notwithstanding, been pretended that these signs were capable of specializing the lesion of such, or such of the orifices, or such valve, and that thus an irregular, unequal, small, intermittent pulse, announced an aortic rather than a bicuspid contraction. A rapid glance at our different patients will show us that this law, which in some cases may be true, is at least not without exception.

In our twenty-one cases, the pulse has been found more or less unequal and intermittent, only seven times, viz: in the following cases.

*Hypertrophy without very notable valvular lesion (Obs. 1.)* Pulse of medium development, unequal, intermittent and irregular.

*Medium hypertrophy. Thickening of the valves. Cretaceous state of the aorta (Obs. 6.)* Pulse intermittent, little developed, but hard and vibrating.



*Considerable hypertrophy. Deformation of the valves. Bicuspid insufficiency.* (Obs. 11.) Pulse sufficiently equal, but intermittent and disproportioned to the force of the subject.

*Double contraction with bicuspid insufficiency and perhaps aortic. Pericardiac adhesions.* (Obs. 13.) Pulse rather unequal and little developed.

*Bicuspid and tricuspid contraction. Bicuspid insufficiency. Slight aortic thickening.* (Obs. 15.) Pulse sufficiently regular, but rather unequal, of medium development and not vibrating.

*Dilatation of the right heart. Aortic vegetations. Bicuspid contraction. Clots in the heart.* (Obs. 19.) Pulse small, unequal, intermittent and irregular.

*Narrowness and a little bicuspid insufficiency. Slight aortic thickening. Clots in the heart.* (Obs. 20.) Pulse rapid, very small and unequal.

Do we not perceive, as a consequence, the intermittence or inequality of the pulse coexisting with the most diversified lesions, from simple hypertrophy to the most complex valvular alterations and sanguineous concretions in the interior of the heart? and as we may be asked if, at least in the seven cases that we have cited, there was not a necessary relationship between the organic lesions and the characteristics of the pulse, we may add that in the remaining fourteen the pulse is noted as preserving its regularity. I will cite in particular among them, the bicuspid insufficiency (Obs. 2), thickening of the aortic valves without insufficiency (Obs. 3), the same lesion with insufficiency (Obs. 4), contraction of the two orifices, with vegetations of the bicuspid valve and a cretaceous state of the aortic valves (Obs. 16), etc.

We must then recognize the pulse as a useful auxiliary, important even in the study of the lesions of the heart; but we should not on this account exaggerate its importance and expect from it information which it cannot furnish, and thus accord to it a degree of confidence, to which other more precise methods may be able to challenge a just title.

I will, nevertheless, make an exception in favor of one quality of the pulse, of which I have yet said nothing; I allude to the vibratory tremor, which the sanguineous wave sometimes presents to us, and that more frequently, it is true, in the arteries adjacent to the heart, than in those more distant. This quality of the pulse coincides very frequently with the causes of the purring tremor (*fremissement catatoire*), in general; or, even in particular with a contraction of the aortic orifice. (See especially our observations 5, 12 and 18.)

Let us pursue our rational exploration. We have noted the pulse of our patient; let us now uncover his chest and examine the precordial region. I designedly say *examine*, for a simple view will furnish us with more than one sign. These signs are *prominency*, *displacement of the point of the heart*, and the *pulsations* of this organ.

*Prominence* of the precordial region is not always a morbid phenomenon. With certain individuals the left mammary region is more developed and prominent than the right, and this anomaly may have been congenital or produced by the oft repeated exercise of the muscles of that side, as for instance occurs with left handed persons. Again, a precordial prominency may owe its existence to a deviation of the spinal column; we perceive, for example, that under the influence of a deviation of this column in which the convexity is thrown forward and to the left, the precordial region must become prominent. But if it be true that this prominence is, in certain cases, without symptomatological value, there is no reason to conclude that it is always so. Almost constantly in pretty extensive hypertrophies of the heart, and in hydro-pericarditis, the more or less circumscribed elevation of the precordial region is an incontestible pathological phenomenon, susceptible even, although rarely, in the course of certain acute maladies of the centre of circulation, of presenting like precordial dullness itself, a sort of augmentation and decline, appreciable during a very short lapse of time.

This precordial prominence, sometimes easily perceived at first sight, is not always so evident. In order to be able, not only to verify its existence in doubtful cases, but also to measure the degree of it, I have caused M. Charriere to construct a small and very simple instrument, which I have called *cyrtometre*. It is a plate of steel, surmounted, on its middle part, by a graduated scale, along which a slide glides, with two blades, parallel to the plate of brass, and fixed to a certain extent. It is understood that this instrument can verify a precordial prominence only on condition of a previous application to the right mammary region, varying its application to many points, and taking great care, in the these different applications, to employ equal pressure.

By the aid of these precautions, the cyrtometre has always appeared to me to fulfill its object properly. Moreover, the precordial region which first suggested the idea of that instrument to me, is not the only point to which it may be applied,

for wherever it may be of practical interest to measure a limited pathological prominence, the cyrtometre may be usefully applied.

*Displacement of the point of the heart.* Some observers have said that the point of the heart beats, normally, about  $1\frac{1}{2}$  inches below the nipple. This localization, true in a good number of cases, does often fail in fact; and why? The mamma is susceptible of very extended congenital transposition, especially in a vertical direction. I have seen the point of the heart at a distance almost double that indicated, the organ being perfectly sound. If we desire to adopt the nipple as the mark, let us take it rather as a lateral limit, and we may then say that the point of the heart beats between the nipple and sternum, nearer to the former, than to the latter: but let us add, as an essential localization, and almost infallible, *in the fifth intercostal space*. This is so exactly true that many years of close observation have not furnished me with a single exception to the rule, and although I have sometimes had reason to suspect the existence of an organic affection of the heart, which nothing caused me to anticipate but that the apex of the heart presented itself more without, or lower than the point I have indicated.

Let us remark that, in the organic affections proper to the heart, it is in both directions or only in one that the displacement occurs. In other cases the morbid lesion is generally seated elsewhere. Thus I have seen pleural effusions of the left side press the heart even into the right mammary region. I have also seen abdominal tympanites or ascites, elevate it as high as the second intercostal space; and that, even, notwithstanding the coexistence of a considerable hypertrophy of the organ, of which our 10th observation has furnished us an example.

Let us add, in fine, that we do not always see the movements of the apex of the heart, and that frequently it is only by a strong pressure of the finger that we are able to perceive them.

This reflection is not less applicable to the third sign, of which we are about to speak, and which I have indicated as capable of being presented to us by an inspection of the precordial region. I allude to the pulsations of the heart itself.

With an emaciated subject, and with whom under the influence of any cause whatever, the contractions of the heart are accelerated, we will see the precordial region elevated, not only by the point, but also, although more feebly, by the base

of that organ. This elevation may be more or less abrupt and strongly pronounced; but, save in morbid cases, it will be little extended, generally circumscribed in a surface of from  $2\frac{1}{2}$  to  $3\frac{1}{3}$  inches square, at most. In organic affections, on the contrary, these pulsations may be much more extended; they may possibly be felt to an extent almost double; they may even propagate themselves by means of the diaphragm, to the pit of the stomach, at the same time that their impulsion raises abruptly and visibly the stethoscope, or even the head of the observer. The extent of these pulsations is here the principal fact; their force is only a secondary consideration; this impulsion, unless it exceeds certain limits, which practice teaches us to recognize, being compatible with the physiological condition. Every one knows, in fact, under the influence of rapid movement, the heart *beats strong*, as it is vulgarly termed, but it does not beat on this account with a proportionably augmented extent. These expanded pulsations constitute then a morbid phenomenon, and I can say, that they characterize essentially, a peculiar state of the heart—hypertrophy. The same is true of the preceding signs. As for these, besides the existence of this last sign, (expanded pulsations,) it is not so necessary in hypertrophy that its absence must immediately exclude the idea of an affection which its presence characterizes. This sign may fail more or less perfectly, and that in consequence of the embonpoint of the patient, or of the coincidence of some other affection, such as effusion, either pleural or pericardiac; or even in consequence of the feeble degree of the hypertrophy or the limited energy of the ventricular contractions, enfeebled by the progress of the malady or the medication employed to combat it.

May I also say that if hypertrophy of the heart is characterized by the extent and vigor of the pulsations, that the absence or slight degree of energy of these, may be owing to an opposite condition, that is to say, atrophy, a condition very rare, and which the preceding observations have not presented us an instance; this condition of the heart, contrary to hypertrophy, existing most frequently above, and even supposing, almost necessarily, the absence of all valvular lesion?

Finally, when it is by the application of the hand that we appreciate the pulsations of the heart, a special peculiarity may offer itself to our observation; I allude to a certain sensation of restraint, or embarrassment, in the ventricular con-

tractions, a sensation difficult to describe, and which, I confess, requires for its perception a high degree of practice, but which expresses then, to the experienced hand, the existence of pericardiac *bridles*, or adherences, which obstruct the movements of the heart. I will refer, in support of this remark, to our 13th observation in particular.

If the anormal extent of the pulsations of the heart, in the strongest marked cases, may sometimes conceal itself from our sight, or escape the perception of the hand, there is another mode of exploration from which hypertrophy cannot, in general, remain latent; I allude to percussion.

Normally, the *dullness* which corresponds to the presence of the heart is from  $1\frac{1}{2}$  to  $2\frac{1}{2}$  inches square, when this dullness exists. I add this reserve; for very often, percussion, the most cleverly practiced, will not encounter a dull sound in the precordial region; it may be that the stomach distended with gas, makes, by the clearness of its resource, the obscurity of the sound that you seek; or, that which is most common, a portion of the lung may interpose before the heart, and of sufficient thickness to produce the same effect. In morbid cases, on the contrary, and especially in rather notable cases of hypertrophy of the heart, percussion not only verifies, but also measures, and that often with mathematical precision, the enlargement of the organ. There is but a small number of clinical facts where this mode of exploration has been in default: and we have seen in two cases, among others, how exactly its teachings may conform to the results of the autopsy (see observations 7 and 21). We have seen that it was especially this anormal dullness which served as data for estimating the weight of the heart, a secondary part of the diagnosis, yet often confirmed after death. The limits of this dullness have varied in our observations between the normal, and 6 inches in one of its diameters, (Obs. 19.) Generally it is in the transverse diameter that we have found the extent of the dullness most considerable.

Up to the present, and in all that which preceeds, we see only some signs of hypertrophy. Let us come now to some more profound lesions. And first, if we always suppose that one of our hands is applied on the region of the heart, a new sign may yet present itself then to our observation. I allude to *vibratory tremor*.

This phenomenon, signalized for the first time, by Corvisart, supposes necessarily, or at least most generally, as well as most of the morbid sounds of which we will speak presently, two conditions;



1st. A certain degree of quickness and of volume in the sanguineous wave, and as a consequence a certain force in the ventricular contractions.

2d. An obstruction, more or less powerful, which imperfectly checks the passage of the blood and augments the collision of its particles. Let us remark, in anticipation, that we will find these two same elements in all our bruits de souffle. Thus then if it is not true that all souffle supposes necessarily a vibratory tremor, it is incontestible, on the other hand, that all vibratory tremor necessarily supposes a bruit de souffle.

Vibratory tremor is not a rare phenomenon. In our twenty-one cases, we have encountered it six times (Obs. 12, 13, 16, 18, 19, 21). If now we inquire, what are the lesions with which it coexists, we will perceive that in our observations, they were constantly either fleshy vegetations or concretions at one of the left orifices, or more frequently at both.

M. Bouillaud says that there have been some inexplicable cases of vibratory tremor of the heart without organic lesion. If, in this respect, the sign of which we have just spoken can sometimes be equivocal, it will not be thus with those whose history I am now about to commence, or rather, resume, and which is entirely auscultatory.

*Sounds of the Heart.*—The stethoscopic minutiae into which we entered, in relating each individual case, and the summary considerations with which we have prefaced our exposition of facts, might perhaps excuse us from the performance of our remaining task. I believe, however, that the collecting at present, of all that these practical reflections may offer of a general character; that the gathering together of these disseminate traits, and uniting them in a table which resumes them, or which may develop, will be adding to these diagnostic studies, a useful at least, if not a necessary complement, the clinical interest of which may cause to be forgotten our repetitions and vicious composition.

I divide into six principal categories, the diverse sounds perceived by our ear when we auscult a heart. These sounds are *normal*, *super-normal*, *sub-normal*, *modified*, *replaced*, or *accompanied*. Let us rapidly run over these six divisions.

1st Division.—*Normal sounds of the heart.*—I will not here repeat all that I said at the commencement of this work, upon the four causes which seem to me to contribute to the production of these sounds; upon the predominance of valvular

clacking which our clinical studies have since so fully demonstrated; upon the anatomical reason for the first sound being deeper and longer, and the second, clearer and shorter, etc. I will only recall that of these two sounds; the first is heard in its maximum below, and a little without the nipple, and the second above and within, at the same time acknowledging that this difference is often more easily seized in the morbid than in the normal state; and I will advise the student who desires to improve himself in the diagnosis of the diseases of the heart, to auscult carefully, and for a long time healthy hearts before ausculting diseased ones. Indeed, one would scarcely believe that the normal heart could be susceptible of slight differences so various and so difficult to describe, and how much the knowledge of these various differences afterwards, facilitates the study of abnormal sounds.

One word more, before quitting the normal condition, or a stethoscopic peculiarity which I have not seen pointed out anywhere, of which I think it is well to be warned.

It has happened to me, rarely it is true, only three times during my two years of *clinique*, to hear at the moment when I applied my ear to the precordial region, a confused buzzing sound, which at first, I did not know how to explain, when, after having continued so long that I thought it my duty to note it in my observations, it disappeared gradually and did not return. This buzzing appeared to me to be a rumbling sound, (*bruit rotatoire*) resulting probably from some febrile contractions of the pectoral muscles; and in fact, the third time that I encountered it (it was in a patient attacked by apyretic gastro-intestinal embarrassment) after having uselessly essayed the interposition of the stethoscope, the suspension of respiration, etc., I caused the patient to be seated at ease on the bed, the arms supported, and almost immediately the sound ceased, to reappear no more, and permitting me then to distinguish the sounds of the heart, which were perfectly normal. Of my other two patients, one was affected by a cerebral hemorrhage, in process of resorption, the other was a woman with a mixture of hemorrhage and softening of the left hemisphere, with partial meningitis. I inquired of myself if the rigidity and contraction of the left superior extremity was not a reason for supposing some analogous and profound contraction of the precordial muscles. But it was not long before I perceived that this was not a valid hypothesis, when I discovered the rigidity of the arm persisting and my

rotatory sound disappearing. It would be useless to say that at the autopsy I found nothing special about the heart.

Besides the three facts that I have cited, there was another example of the same sound in our observation 21 (see above). I have specified it in this instance, however, only with doubt, having been simply editor of that observation which dates, as the reader may perceive, at an epoch much anterior to that of my entrance into the service.

But I have said enough, I think, of a phenomenon essentially accessory. Let us come now to the morbid sounds, properly so called.

2d DIVISION.—*Sounds super-normal.*—The sounds of the heart may surpass the normal state, if I may thus express myself, in relation to their clearness, their fullness and force; in a word, they may be clearer or fuller than normally.

The exaggeration in the clearness of these sounds has not always a very positive clinical value. It may be owing to the thickness of the pectoral parietes, or to the proximity of the stomach distended with gas, as is very often observed with chlorotic girls. Sometimes, however, it is caused by a dilatation of the cavities of the heart, especially if this dilatation, coincide with emaciation of the parietes (passive aneurism of Corvisart) a rare lesion, considered as coexisting with that complete absence of valvular deformation, that the sound of which we speak, suppose. It is more common in a heart organically affected, for one of these sounds only to present us this character of clearness, and then most frequently it is the second. We conceive that this last being naturally the smallest of the two, however little the first sound may be observed pathologically, by this fact alone, the contrast between them will become sufficiently striking to astonish our ear.

The *stronger and well struck* sounds, as they are sometimes termed, are not incompatible with the physiological condition, or at least with the absence of all organic lesion. As a consequence of rapid motion, in some cases palpitation, purely nervous, the heart often beats with sufficient force for the sounds to feel necessarily the effects of the vigor of the impulsion. If this be true in cases where the force of the ventricular contractions is only momentarily exalted, it is so, for a stronger reason, where the exaltation is permanent and organic—in cases of hypertrophy. But we will then rarely be able to note the sounds of which I now speak, for however little the valves partake of the hypertrophy of the fleshy tissue itself, a new shade immediately presents itself and the sounds

are *dry* and *parchment* like. I will refer, for example, to our 1st observation, in which the sounds were described and where the autopsy showed us such a feeble thickening of the valves that we noted it as a case of almost simple hypertrophy.

3d Division.—*Sub-normal sounds*.—In this division, diametrically opposite to the preceding, we may admit two subdivisions: the sounds may become simply enfeebled or entirely null. We should remark, of course, that in the latter event, two conditions are possible: the heart may cease to beat, as for example in complete syncope: or, its beatings may be distant from the ear and separated from it by a liquid—a feeble conductor of sounds. We know that in a considerable hypertrophy, the absence of the sounds of the heart is not a symptom less characteristic than precordial prominence, the extent of dullness, etc.

If the sounds, instead of failing entirely, are only enfeebled, that is to say, if they are less *thrilling* and *not so strong* as natural; if they are, as they are then termed, *heavy*, *hoarse*, *veiled*, or *distant*, different conditions, either morbid or physiological, are possible; thus, to begin with these last, the embonpoint of the patient, the interposition of a thick lamina of the lung or a semi-syncope, are so many circumstances that necessarily effect an enfeeblement of the sounds of the heart. And let it not be believed that this is a puerile remark, without practical value: I have very often seen cases where even a practiced ear was permitted to hesitate in the interpretation of the sound of which we speak, between one of the circumstances which we have cited, and a lesion that we are now about to particularize.

These lesions are very various, and as one may readily conceive, many of them are, in some respect, indicated in advance by the category of the opposite cases, that we have studied above.

Thus if the dilatation of the cavities of the heart, with emaciation of its parietes, may exaggerate to our ear the shrillness of the sounds of that organ, it is evident that sounds entirely opposite must be the result of opposite anatomical conditions, the thickness of the walls of the heart coinciding or not with the contraction of its cavities; if simple hypertrophy manifests itself by sounds forcibly struck, atrophy is expressed by sounds feebly marked.

But further, and it is the first time since we spoke of the auscultation of the heart, that the play of the valves, so important in the stethoscopic history of that organ is presented

to us, the beginning endocarditis sometimes enfeebles the *valvular* sounds—renders the valves *hoarse*, if the expression may be used ; as if, in this respect, there existed a sort of symptomatic analogy between the slight tumefaction of these membranous veils and that of the vocal cords, the first indication of a laryngitis.

We have shown, in the course of our clinical analysis, how the blood, cut by the tendinous cords of the heart, sometimes leaves a fibrinous deposit on the interior of that organ, and especially on the tendons themselves, or on the valves into which they are inserted. This coagulation, molecular in its principle, this first rudiment of fibrinous concretions, which may become sufficiently voluminous to completely obstruct the play of the heart, also produces a partial extinguishment of the vibrations of the valves and deadens their clacking.

Finally, before a hydrops pericardii becomes sufficiently abundant to smother completely the sounds of the heart, we perceive that the first effect must be a gradual enfeeblement of them, so that they are heard as if at a distance, which is, in some sort, the prelude, more or less graduated, of their complete effacement.

4th Division.—*Modified sounds of the heart.* This modification may have respect to the *seat* of the sounds, to their *tone*, to their *rhythm*, and to their number.

1st. *Sounds of the heart modified in their seat.*—This subdivision corresponds to the displacements of the heart, may be either congenital or acquired. We know that in congenital displacements of the heart, the other organs contained in the thoracic and abdominal cavities, present the same transposition. As to acquired displacements, they result almost exclusively from pleural effusions. I have seen in our wards. (St. I de Dieu) a patient with whom, under the influence of this cause, the heart beat immediately beneath the right breast. I have seen, with another, the heart situated in the region of the inferior hollow of the sternum. In these different cases, and the first itself, as remarkable for the displacement of the heart, would justify us in believing that the diagnosis would not at first find itself indicated by the complete absence of the sounds of the heart in the precordial region. This would be an error. These sounds, on the contrary, are often distinct at that point, owing to the propagation, that I have seen, more than once, deceive the ear. momentarily, at least, and until a more prolonged exploration had exactly ascertained, sometimes by auscultation alone.



and at others by the inspection and application of the hand, the anormal point of departure of these sounds. Now, I specify this subdivision only as a memorandum, and it is evident that it ought not to detain us here.

2d. *Sounds of the heart modified in their tone.*—It may be thought that this subdivision is a repetition of those relative to the augmentation or diminution of the clearness of the sounds of the heart, if I did not define the terms and exactly precise the spirit of this new subdivision. When I say that the sounds of the heart may be modified in their tone, I do not intend to express an impression, in relation to either an excess or defect of one of the qualities of the sound perceived by our ear; I allude to a new quality, a modification of the sounds of the heart, that M. Bouillaud designates under the name of *dry* or *parchment* like sounds: not, as it is sometimes believed, that there is the least relationship between this shade of sound and the parchment or new leather sound, which we will find hereafter; but because to an ear, practiced in auscultating the heart, the sounds then present a character of dryness and hardness, analogous to that of the clacking of parchment valves.

I do not doubt but the reader, as yet but little accustomed to our method of explanation, will consider this remark rather too minute, perhaps presumptuous.

I acknowledge on my part that time and long experience were necessary to enable me to comprehend the clinical value of this denomination, and especially to familiarize my ear to a shade of sound, sometimes so difficult to appreciate. But, I may add, that to me now, this morbid sound is one of the most incontestible that I know, and the expression *bruit parchemine* one of the most correct in our stethoscopic vocabulary.

I will say more; the sound of which we are now speaking is, in my opinion, one of the best reasons for relying on the theory of M. Roannet. And, in fact, the lesion to which this sound corresponds, a lesion constant and unique moreover, is nothing else than a thickening of the valves, without deformation; neither is this slight puffing, the result of a commencing endocarditis, which we have seen above produce a hoarseness of the valvular sounds, but a little more induration without doubt, in these membranous laminæ, a little exaggeration of their nutrition; in one word, not a congestion of the valves, but rather their hypertrophy. I will refer, for examples, to our observations 1, 7, 10, etc.

3d. *Sounds of the heart modified in their rhythm.*—We have seen above that the pulse was susceptible of becoming unequal, irregular, and intermittent, which well understood, have their point of departure and cause in a sort of corresponding ataxy of the contractions of the heart. We should expect, then, to find, in these cases the same disorder in the valvular sounds. And, in fact, these sounds may offer to us, in their succession and in their rhythm, quite as many analogous disorders. They may even offer them yet more numerous; and the reason of this difference is very clear. In order that the radial pulsation, that kind of distant echo of the ventricular systole, may be perceptible under the finger, it is necessary that there should be at the same time a certain volume of the sanguineous wave and a certain force of impulsion on the part of the heart. We conceive that these two conditions are necessary in a less degree, in order that the result nearest to the systole or even, to the arterial diastole, that is to say, the readjustment of the valves, may become sensible to our ear. Then we will be able to perceive a greater number of valvular sounds, normal or not, than we can count radial pulsations with the fingers. It is that which takes place, for instance, in what M. Bouillaud has named the *faux-pas of the heart*, that is to say when the ventricle, incompletely filled, as a consequence of an auriculo-ventricular contraction, beats whilst almost empty. It is that which takes place in false intermittence of the beats of this organ, that is to say when, instead of being completely absent, as in the true intermittence (*stoppage* or *hesitation* of the heart,) the ventricular systole is only more feeble. It is the same that may happen in those disturbed and ataxic movements of the heart, when its beats of unequal force, and repeating at intervals also unequal, present to the ear nothing but confusion, tumult, and, if I may thus express myself, a chaos so much the more inextricable at times, as it is not rare for the two times to participate and that thus for a single systole there are two or three diastoles, as, in other instances, for a single diastole there may be two or three systolic movements.

Disturbances, so pronounced as these, coincide, we may say necessarily, with one or more organic lesions of the heart. We have shown in reference to the pulse that they cannot furnish us any positive indication of the species or special seat of these lesions. I will now go farther; there are some of these disorders which may coexist, with a complete ab-

sence of these lesions, with palpitations, the result of causes, purely nervous. I have been more than once warned, by the defect of the rhythm of which I speak, of the existence of this sort of palpitations with patients who did not care to inform me in reference to them. In these cases it is in general the intermittence that we observe; sometimes also there are other irregularities, but which seemed, at least, to be subjected to a certain order of periodicity, being reproduced, for example, in the state of the usual calm of the patient, often nearly a uniform number of regular contractions. Now, there is only one secondary consideration in the differential diagnosis of these nervous irregularities and those that depend on organic lesions. The latter are always accompanied by a train of other symptoms, such as the abrupt, but otherwise vigorous and massive impulsion of the point of the heart; such as souffles and other morbid sounds, etc., symptoms, the absence of which characterises the palpitations which result from the neuroses.

4th. *Sounds of the heart modified in their number.*—In ausculting the heart we sometimes hear three and even, but more rarely, four sounds instead of two. This peculiarity is rather curious than of any very notable clinical importance. It is not rare, at least for the triple sound. Thus in the wards of M. Bouillaud I have sometimes seen two or three examples of it at the same time. In almost all the cases, that I have observed the anormal duplicity existed at the second time, and the two last sounds were sufficiently approximated and brief, in relation to the first, to remind us of the dactyl of the Latin verse, composed as every one knows of one long and two short. It is this triple sound, this *bruit dactyloïde*, if I may be allowed the expression, that M. Bouillaud has compared to the roll on the drum, or to the triple sound of a hammer that rebounds on an anvil.

It is with organic lesions of the heart only that this modification of the rhythm of the sounds has been observed to coincide; but with what particular lesions? This we are not able to determine in a very positive manner, M. Bouillaud, in his *Treatise on the diseases of the heart*, says that he has never encountered the rhythm of three or four sounds, but with patients afflicted with contraction of some one of the orifices of the heart, with induration of the valves, accompanied ordinarily with the sequences of pericarditis. More than once, at the bedside of the patient, he has appear-

ed to us disposed to refer the triple sound to a contraction of the bicuspid orifice, with hypertrophy of the left auricle, supposing, without doubt, that the auricle contracted twice on the sanguineous wave intercepted by the narrowness of the orifice, whence resulted the third sound, which was necessarily, (and in fact it is what ordinarily occurs in the triple sound,) a souffle quite short. I have among my observations only a single case of triple sound submitted to an autopsy. I have cited it above (Obs. 21); we have there seen a contraction of the bicuspid orifice and a hypertrophy of the left auricle. I have seen this same special hypertrophy existing in the heart of a horse, with which Dr. Leblanc had many times verified a very distinct triple sound. These, then, are facts favorable to the explication that I have given. But, on the other hand, how often have we seen this hypertrophy of the left auricle, with contraction of the bicuspid orifice, and our own observations have furnished us more than one example of it, unaccompanied by supernumerary sounds. Can we say that the quickness of the circulation has been the sole cause of this absence? For my part, in agreeing that there have been cases in which the triple sound was really produced by the cause that we indicate, the fourth sound appears to me to have been sometimes also the result of pericardiac friction, which was added to the sound of the first time, I prefer to say, (and M. Bouillaud is not far removed from this idea,) that these disorders in the rhythms of the sounds of the heart are the effects of a corresponding disorder in the synchronism of its usual sounds, or in their relative duration, a derangement that may be owing to a lesion of any one of the orifices. Let us suppose, in effect, that at the second time, for example, the clacking, normal or not, of the aortic or pulmonary valves having ceased, a little souffle still continues, produced by the retarded passage of a jet of blood restrained by the narrowness of an orifice; evidently this orifice may be either the bicuspid, contracted, or the aortic, insufficient. Let us now suppose, and I have seen examples of it, but more rarely, that the doubling of the first time may be the cause of the triple sound, it is still evident that this doubling of the first sound may be the result of insufficiency of the bicuspid orifice, (against which a little bloods till rubs when the clacking, more or less normal, of its valves, has ceased—a clacking not incompatible with its insufficiency,) or the tricuspid clacking; oftener still it will be the result of contraction of the aortic orifice whose souffle, it will be readily perceived, may



be more prolonged than the bi or tricuspid's clacking—or finally, that the left ventricle, contracting at two efforts upon the column of blood incompletely expelled, the bicuspid valve thus clacks twice immediately and successively.

It is well understood that, in order that this doubling of the times may be perceptible to our ear, it is necessary that a certain interval exist between this double time and the return of the other; it is only necessary that the rapidity in the succession of the sounds should not be so great as to reproduce the simple sound before the double may have been perceived. This remark, suggested by reason, is moreover proved by observation.

There is, then, no difficulty in accounting for these multiplied sounds, and in showing that they weaken in no respect our valvular theory. I will go farther; in reflecting on the possible if not efficient duplex conditions of sound, which naturally prevail at the production of each of the sounds of the heart, that is to say, the simultaneous escape of the blood by one orifice, and its arrival by another, we may be astonished that the triple or quadruple sound does not exist more frequently, if we do not remember the silence that exists between the two sounds that I have just pointed out.

In opposition to this multiple sound, M. Bouillaud has remarked that there is often, on the contrary, unity of the sounds of the heart, a unity, if not real, at least apparent and depending upon a two-fold circumstance:

1st. The existence at one of the times of quite a prolonged souffle.

2d. Brevity of the interval that separates the two times.

In these cases the souffle absorbs, in some sort, the other sound, and does not permit it to be appreciated, because it either still continues or is repeated when the other sound is produced. We have, in the observations above cited, specified many examples of this fact. We have noted that we may often be wrong in supposing the existence of a double souffle, and that a good precaution, before settling upon this idea, consists in removing the ear from the precordial region, in order to ascertain if the moment will not occur when the souffle, enfeebled by distance, will not permit the clacking of the other sound to be distinguished. We may, but less promptly, arrive at the same result, in retarding the movements of the heart by means, for instance, of digitalis. By this means the interval between the two sounds is extended, and they are rendered distant from each other.



There does, nevertheless, appear to exist some cases in which the second sound really fails, the movements of the heart being extremely feeble. I have observed no such facts. But M. Bouillaud specifies them, and adds that, well understood, the absence of the second sound supposes only a feebleness and not a complete failure of the corresponding movement of the heart—a movement without which we cannot conceive of the circulation.

5th DIVISION.—*Replaced sounds of the heart.*—In all the morbid sounds that we have passed in review up to the present point, it was possible to find and to recognise, more or less clearly, the primitive character of the normal sounds of the heart—the valvular clacking. In one word, all these sounds might be considered, with more or less reason, as altered clacking. In those which are the object of the section upon which we now enter, there is no more clacking, or at least, the clacking, if we still find it, will be no more than an accompaniment—an accessory often itself unnatural, a sound altogether of a different nature, both in relation to its sonorous qualities and to its cause.

And, in fact, as we have already said, the valvular clacking is the result of the readjustment and of the vibration of the valves of the heart: this new sound proceeds most frequently from the fact that the valves are no more able to readjust themselves or to vibrate properly. In the clacking there was a shock of blood against a valve: in the sound of which we are about to speak there is friction of the blood against this valve. Clacking indicates to us only that an orifice closes: this sound indicates in general that the orifice is not closed. Clacking has its point of departure only at the orifice through which the sanguineous wave passes; this sound has its point of departure sometimes in the same orifice badly closed, but more frequently at that through which the blood escapes, and which is imperfectly opened. The seat of the first then is single, that of the second may be double. (It is understood that I speak here of only one of the valves of the heart.) I mean to say that the indications of the second will be less easily gathered and less precise at first than the semiosis of the first. Every one has, doubtless, divined this morbid sound; it is the *souffle*. The *souffle*, primitively called *bruit de souffle*, comprehends all the *valvular* sounds that now remain to occupy our attention. The veritable proteus of the stethoscope, it is it which under the influence of such or such modification in the mobility of a valve, in the contrac-

tion of an orifice, let us rather say, under the inspiration, more or less happy, more or less poetic, of the observer, that I say we will see it transform itself now into the sound of a rasp, then into that of a file or a saw ; now flexible and soft, anon rude and creaking ; sometimes chirping like a young chicken, at others warbling like a turtle-dove, or yelping as a little dog, etc. Let one not believe that all this is but a sport of the imagination or unnatural metaphors ; no, they are, on the contrary, only faithful translations of impressions really perceived—the veracious and positive interpretation of a sound observation.

Are we to conclude, hence, that the *souffle* is a sign easily appreciated, which requires only a slight attention, and that it strikes the least practiced ear ? This would be an error ; for, more than all others, perhaps, this morbid sound offers to us shades and degrees, we may almost say, indefinite. But to speak first of the best characterized *souffles*, of those raspy sounds, if you will, or of the cries imitating those of certain animals, these do generally, doubtless, present themselves readily to our ear. But who can tell if they occur in the heart or in its envelop, if their seat be not in the pleura itself, of which the movements are not always so easily suspended as some think ? Who can tell even, in some cases at least, to which of the two times of the heart the sound in question corresponds ? And if it is permitted sometimes to hesitate thus, when at least we are assured of the existence of this morbid sound, how will it be with those light, rapid, fugitive *souffles*, which we must seize on the wing, if I may be allowed the expression, and to localize the same ; that one may so readily allow to escape, or may confound now with a slight grazing of the pericardium, and then with a pleural friction, and who knows but sometimes even, in a too hasty exploration, with one of the vesicular sounds.

I cannot impress too strongly on those who desire to study the maladies of the heart attentively and conscientiously, to exercise the ear to the *bruit de souffle*, and, as I have remarked above, the best preparation for this is the frequent auscultation of the normal heart. And, in the second place, and when once the ear knows well what a *souffle* is, and of what principal shades this morbid sound is susceptible, it is necessary to know how to recognise the special time to which it corresponds ; if it is simple or double ; if it is really or only apparently double ; if it does or does not mingle with valvular clacking, or friction of the precardium, or pleura, etc.

Thirdly, and finally, and it is not the point most easily determined, it is necessary to ascertain to what point of the precordial region the maximum of the souffle, is to be referred ; is it heard most distinctly without or within the nipple ? Is it towards the point or towards the base of the organ ; or yet towards the left border and in the inferior hollow of the sternum ? Finally, does it or not propagate itself into the ascending aorta and into the carotids ? We are about to see that each of these indications has its peculiar value and diagnostic bearing ; or rather, have we not already seen it many and many times in our clinical analyses ? It is here especially that I would have cause to fear very frequent repetitions if I had not imposed on myself very contracted limits, and if I did not remember that I ought to resume now rather than to develop the practical considerations which attach themselves to our subject. Let us then study the souffle succinctly, in each of the times of the heart isolated, and also in the two.

1st. *Souffle of the first time.*—We must acknowledge that this souffle is often, by itself, one of the most insufficient signs that we have yet encountered. In truth it may exist with or without organic lesion of the heart. In the first case, the cause may be simply an increased rapidity of the sanguineous column ; I have spoken of this fact, in the commencement of this memoir, with sufficient details to obviate the necessity of recurring to it here. But most frequently it is accompanied by special alteration of the blood—chlorosis or chloro-anemia.

By what characteristics shall we recognize the chlorotic souffle ?

By the following ; like that produced by quickness, this souffle never exists but at the first time ; it is generally very slight and short ; it *accompanies* the clacking of the first rather than masks it ; it is heard especially in the region of the aortic orifice and very often exclusively at this point ; sometimes, in truth, it is propagated along the ascending aorta, and now and then as far as the carotids, but in these cases, it is stronger and not so brief as I have just supposed it ; and, in fact, (I speak only of chlorosis, properly so called,) it may be sufficiently prolonged, intense and even rude enough to simulate a souffle of organic origin : in fine, it is very often inconstant and variable in respect to its intensity and even in regard to its existence ; such a souffle, now very distinct, may immediately become entirely inappreciable.

What is the determining cause of this souffle? Let us seek, in the characters themselves that we have assigned to it, if we cannot encounter some of the elements of this problem. We have said that the maximum of this souffle was heard in the region of the aortic orifice; thus, then, is its seat already anatomically fixed. It exists at the first time; then it is at the moment that the sanguineous column passes this orifice, that it is produced. But, as we have seen, all species of bruit de souffle suppose an excess of friction; to what then can this exaggerated friction be owing? Evidently we should seek for the reason only in an anormal condition of the sanguineous column, or of the aortic orifice. The condition of the blood should first receive our attention; for every one knows that it is precisely the grave modification that this fluid undergoes in its composition that constitutes the most essential element of chlorosis. Certain observers have dwelt upon this point, and they have said: Physics teaches us that the friction of a fluid is in an inverse ratio to its density; then blood becoming less dense, such as chlorotic blood, ought to produce more friction; but they have not anticipated this objection, that if it were thus, the friction, and by consequence the souffle, ought to cease only when the blood shall have resumed its normal density, and that this fluid does not momentarily vary in its composition, whilst however the souffle in question has an inconstancy and variability but little compatible with this fixity of chemical constitution. There remains then the aortic orifice. Do we not find here some possible conditions of narrowness, of contraction, transient, variable and intermittant? Yes! if we consider on one hand, in virtue doubtless of the old adage: *sanguis frerat nervos*, chlorosis is a disease remarkable for infinite troubles or disorders, essentially nervous; and on the other hand, that by means of the ramifications of the great sympathetic, which distributes itself to their coats, there exists between the arteries and the nervous system the most intimate connection, a very frequent cause of arterial disorders essentially local. Hence those flushes of facial heat, hectic flushes, those circumscribed pulsations now of the carotids and anon of the cœliac trunk, etc. Hence those anomalous arterial contractions, those spasms, those contractions semi-convulsive, variable and very extended, like the apparent and external phenomena that I have just cited, and like all that, moreover, which is owing to a nervous ataxia, and which will produce here, the bruit de diable and its numerous variations; there, a simple souffle, acoustic phe-

nomena so evidently in proportion to the calibre of the vessel in which they are seated, and of which the shades of difference are so very dependent, upon the greater or less contraction that the tunics of these vessels may undergo.

In our opinion, then, the chlorotic souffle is nothing but a nervous spasm of the aorta. It is the bruit de diable of this large vessel—bruit de diable which does not generally go beyond a bruit de souffle, but which however sometimes, in cases of chlorosis of the highest degree, of which I have myself seen an example, may extend to a musical whizzing; this souffle moreover tending generally to become sibilant, there is a coexistence of chlorosis and organic lesion, a cause itself of a permanent narrowness of the aortic orifice.

If I insist somewhat in detail on this chlorotic souffle, it is because the observations that we have reported, essentially consecrated to the study of the symptomatology of the organic affections of the heart, offered us no occasion to particularize it, and that however we might pass in silence a sign which so nearly affects the study in which we are engaged, a sign which may often present so high an interest in reference to the differential diagnosis.

We can scarcely believe, in fact, how many errors in diagnosis this souffle daily causes, how many anemic women, or young chlorotic girls, in whom we every day see combating by repeated vene section, an imaginary affection of the heart, diagnosticated principally from this fatal bruit de souffle, of which the treatment itself becomes then, in some sort, the best aliment.

We understand this error when we see it so often renewed, or when we know how little habit of auscultation so many practitioners bring to the exercise of their profession. But what I cannot comprehend—and I acknowledge that it is revolting to common sense—is, that there are young writers, so very badly instructed, as to accuse of a similar blunder, who? M. Bouillaud himself; M. Bouillaud who has so often pointed out this source of error; these gentlemen tell us seriously that the pretended lesions of the heart, which, according to this professor, coincide with rheumatism, are only simple cases of anemia, and that the souffle which diagnosticates them is only a souffle of impoverishment of the blood, the result of the medication to which the patient has been submitted.

But we will not dwell any longer on an accusation, all the shame of which must recoil upon those who have raised it;



we will now pass to the soufflé of the first time with organic lesion.

Contrary to that of which we have spoken, the organic soufflé, located in the first time, is generally more or less prolonged; it may replace entirely the valvular clacking, which may not really exist, or may not be distinguished from the soufflé that makes it; like the preceding, its maximum may exist in the aortic region; but contrary to it, it may also respond more especially to the bicuspid orifice; like the preceding it may be prolonged into the ascending aorta, and along the carotids; contrary to it, it does not momentarily vary, and especially does not entirely cease to exist when it has been once verified.

Does this soufflé, separated from that of the other time, often exist? No! in all our observations, I perceived it only in the the third and seventh thus isolated. With what lesions does it especially coexist? In the two cases to which I refer it was with aortic lesions; valvular thickening in the first, and a cretaceous state of the aorta in the second. This last lesion, however, is now frequently expressed by a soufflé of the second time, or better by a double soufflé. As to the bicuspid orifice, we conceive that the exclusive soufflé of the first time very rarely responds to its alteration. For what would then be that lesion or insufficiency? a lesion but little compatible with the free play of that same valve, at the other time, save always the exceptional circumstance of an insufficiency owing to a discrepancy between the valves remaining normal and an enlarged orifice.

As we see it, then, and I have more than once made this remark, the time to which a soufflé responds does but indicate the orifice diseased. Here, however, we may say, empirically, that this will be, ordinarily, the aortic orifice. Now we may be assured of this by the seat of the maximum of the soufflé, and by its propagation into the carotids; the absence of this propagation and a difference of seat indicating to us a lesion of the bicuspid orifice. As to the species of this aortic lesion we may readily comprehend that it will always be a contraction, either absolute or relative. The last will coincide, for example, with general hypertrophy, or with a dilatation of the left ventricle; the first will be occasioned by a thickening of the valves, either by a deposit of cretaceous matter on their surface, the presence of vegetations on their free border, or concretions between their lamina. It is sometimes possible, in the diagnosis, to discriminate more or less exactly one of

the lesions that we have indicated. Sometimes, for example, the limit and acuteness of the souffle gives us, in some sort, the measure of the contraction. It is then, according to the expression of M. Bouillaud, a raspy souffle. We have seen that when chlorosis coexists, the souffle may be sibilant. In all cases of extreme narrowness, the pulse moreover comes to our aid by its smallness and slenderness, if there be vegetations, and especially of a cretaceous matter on the valves, the souffle will be less pure in its tone, harsher, less polished, accompanied often by a vibratory tremor, that, in fact, we have more than once seen in our observations, establish the diagnosis confirmed by the autopsy. If there are clots only (they are rarely located at the aortic orifice exclusively,) the dullness of the souffle, its variations sometimes, the palpitations and dyspnœa of the patient, the character of the pulse, the coldness of the extremities, etc., will develop to us this morbid peculiarity.

I have said above, that the souffle, limited to the first time, is very rare. This remark, in my opinion incontestible in cases of rather ancient organic lesions, will be less so as applicable to forming lesions, to those, for example, which begin under our observation during the existence of some acute malady, most frequently a rheumatism. Often, in fact, we are warned of the imminent peril by a souffle yet slight, and which, for my part, I have seen more than once then existing only at the first time. How shall we discriminate between this inflammatory souffle—this veritable sign of endocarditis, and a chlorotic souffle? To an experienced ear, this souffle has at first very often a special tone—a peculiar stamp which will not permit it to be mistaken. May we then believe in a chlorosis or anemia; if the patient has been but little or not at all bled; if the blood drawn from the vein is rich and openly inflammatory; if it makes more than 5° on the *areometre* of Baume, a limit above which the anemic souffle cannot be produced according to the experiments made in the wards of M. Bouillaud; if this souffle diminishes and is enfeebled, instead of increasing, under the influence of emissions of blood; and, finally, if the patient's face is strongly and generally colored, his pulse vigorous and developed, the impulsion of his heart energetic, all circumstances but little compatible with a state of chloro-anemia, and especially with the production of a souffle from impoverishment of the blood; if, above all, we find the precordial dullness enlarged, the point of the heart displaced, etc.? These are the cases in which some have dared to assert that M. Bouillaud mistook an anemia for an

endocarditis. Shall we say that the distinction, of which I speak, between the two souffles of the first time is always so striking? No! doubtless, I frankly acknowledge it, there are some difficult cases; but what are these cases? They are those, in which the individual copiously bled before our examination, or already debilitated by protracted disease, present at once the palor of anemia and the acuteness of repeated rheumatism, inflammation of the lungs, etc. It may be the case with a young chlorotic with whom one of the maladies that I have cited, declares itself in an acute form. Then, in truth, and only then, will we be permitted to hesitate, at least at first, on the nature of the souffle. Then we cannot have had too long a practice, or too consummate clinical experience to surmount this difficult diagnosis. Then, again, the impartial and attentive observer, who may have long followed M. Bouillaud to the bedside of the afflicted, must admire the exquisite tact and shrewdness in diagnosis of that clever practitioner, the certainty and penetration of his glance.

One more thought before we terminate this long paragraph. Such a souffle may be really simple, that is to say, being actually produced only in one of the times of the heart, and for example, at the first time, and yet may appear to us to be double, if on the one hand, it is sufficiently loud to mark the clacking which is isochronous, and, on the other, is repeated so quickly as not to permit the ear to seize the sound that succeeds it. We have seen that the means of discriminating between this apparent and real duplicity is simply to auscult beyond the precordial region. A souffle propagating itself to a less distance than the valvular clacking, will not fail, if the souffle be single, to enfeeble it sufficiently by distance, to disengage the valvular clacking.

We will now take up the subject of the souffle of the second time.

*2d. Souffle of the second time.*—If we are permitted to hesitate, in regard to an organic affection of the heart, because we only perceive a souffle at the first time, the same uncertainty does not attach to that of the second. Then there is always valvular lesion, an obstacle to the free play of the valves. This first consideration already simplifies, in a remarkable degree, the history of this morbid sound, which is moreover, somewhat sketched in advance by that of the preceding souffle. Who does not perceive, in fact, that like the former, the cause of the second souffle may reside either in the narrowness of an imperfectly opened orifice, or in the insufficiency

of a valve imperfectly closed. It is then, still true here, that the sound to which the souffle responds, no longer indicates the injured orifice ; but, however, with the addition of this remark, which the other souffle has suggested to us, that the insufficiency of a valve being scarcely compatible, save in the cases that I have specified, with a disproportion between the valve and the dilated orifice, with the integrity of the other sounds, which the unity of the souffle supposes ; we will be led then, to infer from a souffle of the second time, a contraction of the bicuspid orifice rather than an insufficiency of the aortic valves ; always, however, proving this inductive, first by an appreciation of the anatomical seat of our souffle, and by absence of propagation into the carotids. As to the particular kind of valvular lesions that this souffle supposes, what is the need of indicating them here ? And what would this enumeration be, but a repetition of the corresponding part of our preceding paragraph, presenting no other difference than a transposition of the two orifices ?

Further, such as we will at present consider it, that is to say, isolated, the souffle of the second time is still more rare than the first. I do not even find it pure from all mixtures in any of our observations. I will indicate, nevertheless, an example of this souffle, if not single, relative to the two times, at least so in reference to the play of the same valve, our observation 6 in which this souffle appears to have been produced by the cretaceous state of the aorta ; and our observation 17 where this souffle, single in the region of the bicuspid orifice, corresponded evidently to the contraction of that orifice, without mixture of insufficiency. We will find even well indicated, in this last case, that character of raspy souffle that we have declared to be an almost certain indication of the degree of the contraction of the altered orifice.

Apart from the sibilant souffle of chlorosis, all the other shades of the first souffle may be produced by this, and diagnosed in the same manner. I will not then dwell on this point ; less still will I insist on the apparent doubling that this souffle may present. All that I have said on this subject, in reference to the preceding souffle is exactly applicable to this.

3d. *Souffle of both times.*—Having studied successively the souffle of the first time, and that of the second, we are prepared to comprehend those cases in which a double souffle is the result only of a succession of these two, each of them coinciding, by hypothesis, with the corresponding clacking, which is only more or less completely marked. Evidently,



in effect, for the history of this fact we can only refer to the two preceding histories. But if this state of things be possible; if, for example, we may have simultaneous contraction of the two orifices, without insufficiency of either; or, reciprocally double insufficiency without contraction; we may say that it is not common. I refer in this matter to our twenty-one observations. Among them scarcely a tenth could give us an idea of this sort of pathological anomaly. Much more frequently the two souffles have their cause in the double lesion of only one of the orifices, or of both, or in the double lesion of one accompanied by simple lesion in the other. I will explain myself by some examples; the double souffle being very common in organic affections of the heart, our observations will furnish them abundantly.

1st. Suppose a single valve be at the same time, sufficiently thickened to effect a certain contraction of the corresponding orifice, and so deformed as to re-adjust itself only imperfectly; will we not then have successively the souffle of contraction and that of insufficiency? This is established by our observation. We think the same result will occur if the valves adhering either to each other, (observations 13 and 17,) or even partially to the adjacent parietes of the heart, (observations 13 and 15,) offer either a narrow passage, unfavorable to the ingress of the blood, or a passage always open, too favorable to its reflux.

2d. Let one of the two valves present this condition of double souffle, the other may also be altered, but in such a manner as to offer only one of the two morbid conditions of which we will speak presently; we will still have a double souffle, so much the more marked, be it understood, as we approach nearer to the orifice principally affected. (See observation 14.)

3d. Finally, for a stronger reason, we hear a double souffle if there be a double lesion of each orifice. Our observation 16 furnishes a beautiful example of it.

We should remember that the clots in the heart may yet powerfully concur in the production of a double souffle, it may be by combining their influence with that of the valves already injured, or in constituting themselves the principal cause of restraint on the valvular plays.

Shall I say, in fine, that very often it is without the heart, in the aorta itself, in the cretaceous state of its internal membrane, that we must seek the cause of the double souffle that now occupies our attention? We have insisted often enough,



in view of our facts, upon this pathological circumstance, and upon the means of diagnosing it, to be content with mentioning it here. (See among others, observation 8.)

Let us close with a last morbid lesion, a possible but exceptional cause of a double souffle. I allude to an anormal communication between the cavities of the right and left heart. Now this case is so rare that I only mention it here as a memorandum.

Such are, if I mistake not, the principal circumstances, either anatomical or semieological, that attach to the history of the bruit de souffle. A last touch is, perhaps, wanting to the picture. We have seen in the beginning that there were some souffles possible without organic lesion; let us now say that we have seen some organic lesions, and even some of those that we have enumerated, which had existed without souffle. How shall we reconcile this fact with all that precedes, and is it not sufficient to put in question the validity of our theory? No, doubtless this difficulty is more specious than real. What is the material physical condition of all kinds of souffle? An excess of friction, as we have said. But in order that this result may be produced, who does not perceive manifestly that there is necessary at the same time, a certain obstacle to the passage of the blood, and a certain quickness of motion in this fluid? Can we consequently be astonished, that, if sometimes the excess of quickness may produce a souffle in the absence of organic lesions; in other cases also the insufficiency of this same quickness may have had for result the absence of souffle, notwithstanding the existence of these lesions? This is, at least, as frequent and quite as simple to conceive of as the opposite proposition; and for my part I have had frequent opportunities to verify its exactitude, at the bedside of the patient. I remember, in particular, one of those with whom the pulse diminished most notably under the influence of digitalis: with him we witnessed repeatedly, the disappearance and return of a souffle of the second time, coinciding with the administration and suppression of that medicinal substance.

What shall we now say, and where, in fact, is the necessity of speaking separately of the sounds of the rasp, of the file, of the saw, of chirping, etc., which we have specified above? Do we not know that all these sounds and all analogous ones that may hereafter be introduced into our vocabulary, can never be any thing more than shades of the same bruit de souffle, in relationship with such or such a degree of valvular indu-

ration or contraction of the orifices? It is, without contradiction, the sole special feature in their history, which would be only an useless repetition of that of the *souffle* properly called.

And now that we have traversed the details of this vast ensemble, can we fail to appreciate all the value of this morbid sound, all the predominance of this symptom? Can we not comprehend that if other signs, like this, may reveal to us the existence of organic diseases of the heart, this alone can enable us to penetrate the inmost recesses, indicate to us the orifice diseased, and show us the lesion; that to it, without any doubt, we are indebted in the largest degree, for our success in diagnosis. How then are we to conceive of practitioners so lagging as to treat, as chimerical and fabulous, the legitimate pretensions of our art in this matter, and who maintain that all is yet to be learned in reference to the special seat of the maladies of the heart? Must we not regard a scepticism so blind, as constituting really a sort of flagrant ingratitude for the beneficence of our epoch? As to ourselves, we are grateful to her for having furnished us thus a means of exploration, which the most precise methods of surgery might itself perhaps desire. Thanks to those of our predecessors, I may also say of our contemporaries, who have endowed the service with so handsome an inheritance, and let us endeavor to honor their memory, not only by paying to their names a merited tribute of gratitude, but above all by making every day new efforts to fertilize and develop in our hands the treasure that they have bequeathed to us.

This brings me to confess frankly, that there exists in these clinical researches a real void, so much the more to be regretted as it is difficult to believe that we shall ever be able to fill it properly. I speak of what concerns the local and precise diagnosis of affections of the right heart. I am aware that it has been said, that these diseases being infinitely more rare than those of the left heart, we may generally, and without much risk of error, disregard its diagnosis as of a lesion constituting in some sort an exception. But independently of the scientific inconvenience that there may be in this omission, reflected upon and empirically calculated, I will make the remark, according to the evidence of our facts themselves, that it is true that they are rare, as primitive and capital lesions, but they are no less sufficiently common as secondary ones; and to disdain their solution is to abandon an element of this diagnostic problem often very worthy of itself to fix the attention of the observer.

To what then can this inferiority of the semieological history of the right heart, as compared to that of the left, be owing? To many reasons, without doubt. Thus first to the less frequency of lesions of the right side, which for this reason must be less known; then because the right being more central, in some fashion in regard to its destination, and at the same time less powerful in its contractions than the left, its valvular sounds must be more distant perhaps and less distinctly struck; finally, because its disorders always coinciding with the more grave disorders of the left heart, the morbid results of the latter readily mark to our ear those of the former.

All these practical difficulties are assuredly grave; but are they insurmountable? I think not. We have ever seen, in the analysis of our clinical facts, that our efforts at diagnosis in that respect have not been altogether unproductive. Let us sum them up then in a few words and ascertain what is the actual state of the science in that respect.

Certain practitioners have said: Divide the precordial region by an imaginary vertical line; the morbid sounds which predominate on the right side of this line, between it and the sternum, will appertain to the right heart, those that predominate on the opposite side of the line will belong to the left. This mode of exploration is far from being infallible. That the distinction upon which it rests may be true as applied to the normal sounds, or when the sounds of the right heart are only modified or replaced, I dare not either affirm or deny. But I can say, with an assurance supported by numerous observations, that when the left valvular sounds are gravely altered, they engross in some part our ear, and exclusively absorb our attention, may be on one or the other side of the line indicated. I will refer, however, to our obs. 13 and 14, in which in fact a *bruit de souffle*, towards the inferior hollow of the sternum, was noted during life, and a lesion of the tricuspid orifice verified after death. But, also, and in opposition to those two cases, I will refer to obs. 15, in which a lesion perfectly analogous coincides with the complete absence of this special *souffle*.

The want of certainty then in this sign, was a reason for seeking in others indications more constant and less unfaithful. These other signs are all more or less the immediate results of functional disturbances by valvular contractions of the right heart.

These disturbances affect then, principally, two great functions, respiration and venous circulation.

If we were permitted to assist at the beginning and to follow all the symptomatic phases of an organic affection of the right heart, I do not doubt but the first disorder would be furnished by the respiratory apparatus, and these by consequence more or less generalized by the stasis of the venous blood. But these things do not occur commonly, and like the lesions of which I will speak are consecutive, sometimes terminal; the same is true also of the symptoms that they occasion, showing themselves only at an epoch more or less remote of the stage of the disease and sometimes only in the last days of the patient.

Now, what I say of the right heart, ought not to cause us to lose sight of the relation, yet more intimate, perhaps, which also exists between the lung and the left heart. This receiving directly the blood that the lung sends to it, cannot be obstructed in the least degree, without this arterial stasis communicating itself gradually to the pulmonary parenchyma. The right heart or the contrary sending the blood to the lung, however little the propulsion may be suspended, the nervous stasis reaches the pulmonary parenchyma through the medium of the vena cava superior into which the veins that have nourished the lung itself, empty, and yet it is perhaps true in consequence of this suspension of the *vis a tergo*, that under the influence of the right ventricle, the venous column exerts upon the movement of the blood which has previously reached the thickness of the pulmonary tissue.

To sum up, then, we have on the part of the respiratory functions direct disorders, if I may use the expression, only in cases of very considerable ventricular hypertrophy, if certain valvular conditions do not intervene to neutralize its influence either by breaking the sanguineous wave (contraction of the pulmonary orifice,) or by turning it from its course (tricuspid insufficiency). These disorders in the lungs, are sanguineous effusions, more or less extensive, hæmoptysis alone, or even apoplexy. At other times on the contrary, and this is more common, there is, as in the preceding cases, cough and dyspnœa on the least exercise, but the lung is no longer infiltrated with blood, which is effused; it is engorged with serum, which speedily accumulates at every point where the venous blood ceases to have a free course, it becomes œdematous. Then, also, there soon supervenes many



other analogous results, produced by the same cause; thus the purple color of the lips, the swelling of the face, the infiltration of the extremities, that often open the scene gradually, invades from below upwards the inferior extremities, then the parietes of the abdomen, and even complicating itself with ascites. Then also the jugulars are distended, and we may note the venous pulse, that almost infallible indication of the retrograde movement of the sanguineous wave, through the imperfectly closed tricuspid orifice. I repeat it, to those who have followed the developement of these different symptoms, added to some of those that indicate to us that the heart is organically affected, as prominence, displacement of the apex of the organ, extent of the dullness, etc. the diagnosis of the lesions of the *right* heart cannot be doubtful; and I remember that in a large number of our observations these were the signs that caused us to admit disorders that were demonstrated by the autopsy.

We should remark, however, that all the signs are only the results of functional disturbances, effects more or less removed from the anatomical seat of the pathological cause. Should we compare, under this view, these signs always indirect, to the signs of the immediate product of that lesion itself, which have the same anatomical seat, if I may thus speak, as that lesion to, the morbid sounds, in a word, inherent to the more or less deranged play of the diseased valves?

However we should note, *en passant*, that these were the signs, par excellence, before the discovery of auscultation. They were the characteristic pathognomonic symptoms of diseases of the heart in general in the school of Corvisart, and many of these constituted, what that illustrious observer called the *facies propria* of the disease, adding that these *facies* were the surest guide of the practitioner; signs of but little fidelity, nevertheless, when we consider them, in that extended sense, since they are, if not exclusively, at least principally affiliated to diseases of the right heart, diseases that are in general secondary, and that in these diseases themselves they appear only more or less removed from the beginning; we may say, that they sometimes fail altogether; witness our 17th case, with which the notable dilatation of the tricuspid orifice and of the corresponding auricle was not indicated by any of these signs.

This digression, rather lengthy perhaps, but which merited



a place in the symptomatological study of the lesions of the heart, has caused us to loose sight, for some moments, of the categorical order that we have pursued. Let us then now return to it and enter upon our last division of the sounds of the heart.

6th Division.—*Accessory sounds of the heart.*—Whatever may be the tone of the sounds of the heart, whatever may be the modifications which they present us, in whatever manner they may find themselves replaced, it can so happen, at the same time, certain other sounds appertaining either to the heart itself or to its serous envelop may present themselves. We are now about to consider especially the accessory sounds, at the same time bearing in mind, that most frequently they coexist with one or several of the morbid sounds which we have just reviewed. One other remark is common to them, it is that they may all be considered as finally reducible to the exaggeration of normal phenomena.

Thus to speak first of the heart itself, normally the point of the organ strikes, during its contraction, the walls of the chest, and this percussion, at least according to our theory, does not produce any perceptible sound, or any that we can distinguish from the valvular clacking which is isochronous with it; but, let us admit that this percussion becomes exaggerated, it may be from its quickness, it may be by the mass of the organ, then two distinct sounds can strike the ear. Sometimes it will be a clear, silvery or metallic tinkle, called *auriculo-metallic*, not to indicate, as I have sometimes seen it supposed, that the auricle contributes to its production, but to distinguish it from the metallic tinkling which has its cause in a lesion of the respiratory apparatus, or because it is almost indispensably necessary, to place the ear immediately against the chest, to perceive it.

This sound is easily simulated in gently percussing the back of the hand, its palm embracing one of the ears of the experimenter. This experiment in explaining to us the production of this sound, at the same time shows us its little symptomatic value. What does it indicate, in effect, except that the point of the heart strikes in a hurried, or in a sufficiently distinct manner, a sufficiently thin pericordial parietes, whose thickness is not of a nature to destroy its vibration

That is to say, and experience proves it, if this phenomenon can take place in hypertrophy of the heart, it is equally pos-

sible in the absence of this hypertrophy and of all lesion. It is then an accompaniment little worthy of interest, in a practical point of view. Let us add, by way of completing its history, that generally it is heard only in the systole. Sometimes, however, but by very rare exception, it is double; when that is the case, we are compelled to admit that the base of the heart has a see-saw motion equal to that of the point.

If the auriculo-metallic tinkling is compatible with a normal state, it is not thus of another sound of which I am to speak, and which is, we may say, characteristic of hypertrophy, of which it often not only assures us the existence, but to some extent even the degree. This bruit is a dull, dead sound, the impression of which often accompanied by a visible, anormal impulsion, announces by its tone and its peculiar characters the hypertrophy of the point of the organ. There is something pathognomonic in the sensation of this sound, which permits us to separate it easily from the clearest bruit de soufflé that we find connected with it, though sometimes it may itself be sufficiently strong partly to mask them.

The pericardium is normally the seat of a serous exudation, which, lubricating the inner surfaces of the opposed leaves, softens them and facilitates gliding motion, so that no perceptible friction sound reaches the ear. But let this serous vapor become suppressed or condensed; let the glutinous surface of the pericardium yield with difficulty to the alternate movements which oppose one to the other; let them become roughened, unequal, shaggy; let them be locked by adhesions, tied by false membranes, there will necessarily result new sound, whose tone is relative to the nature and qualities of their anatomical cause; there it is a single brushing, a gentle rumpling analogous to that of a silken tissue; here it is a sound of rasping or scraping, more or less rude; now again it is a sound as of new leather recalling the analogous variety of pleural friction.

All these sounds are, evidently, only varieties of the bruit de frottement, (friction sound,) they constitute the last morbid accompaniment which we have to notice, an accompaniment which it is not always easy at first sight to perceive. We will devote a few moments to their consideration.

The pericardiac sounds, whatever they may be, are generally perceptible during the two times, but most especially in the first; they are more superficial and nearer the ear than

those of the heart properly so called; they are sometimes accompanied by certain general or local phenomena peculiar to pericarditis. Amongst the first I will recall the febrile condition, the anxiety, the agitation of the patient, &c., and amongst the second the palpitations, the irregularities of the heart, and, under the hand, a certain sensation, either of friction, or of difficulty in the movements of the organ, which we have mentioned in some of our preceding observations, (viz. OBS. 13, 14 and 18.) Sometimes very loud, they can mask the valvular sounds, whether they be normal or anormal. At other times, as when they are reduced to a simple rumpling, they can, on the contrary, escape the most attentive ear, or embarrass the most skilled practitioner, in almost perfectly imitating the bruit de souffle. I am aware that in this last case, the souffle has been supposed to be caused by a concomitant endocarditis; but I know of some facts, which cause me to think that, independent of this complication, a false membrane can of itself give rise to a bruit analogous, if not identical, to that of a souffle, doubtless more superficial, more circumscribed, and propagating itself, if I am not deceived, to a less distance; but in good faith, not capable of being exactly diagnosticated save in a special habitude.

The morbid sounds whose history we have just written, the last signs belonging to the auscultatory domain, end the list of the different methods of exploration, applicable to the diagnosis of diseases of the heart. As we have just seen, these methods are sufficiently numerous. Those furnished by auscultation being, undoubtedly, the most important of them all. I have collected them for the purpose of facilitating their study and recollection, within the limits of a portable table, the habitual accompaniment of this part of my course of diagnosis.\* To it then I refer the reader, as to a succinct recapitulation of the more developed of the principles which we have just reconsidered.

If now we cast a coup d'œil upon the various data furnished by stethoscopic and other signs, must we not acknowledge that of our principal organs, either intern or extern, there is not one whose diseases are revealed to us, both generally and minutely by not only so numerous but by such precise signs, that the heart is at the present time one of these organs whose diagnosis is most positive and

\* Synoptical table of the sounds of the heart.

most complete? This conclusion would doubtless appear, at first sight, very rash and presumptuous to certain practitioners, and such that I should have been cautious about publishing in the commencement of this work, but which I now feel quite sure none of my readers will desire to gainsay.

Let us now pass to the third part of this memoir.

### THIRD PART.

#### *Practical application of the Diagnosis of Diseases of the Heart to their Treatment.*

Though the diagnostic result of our clinical researches is well calculated to satisfy an observing lover of his art, there exists an objection to the practical value of these researches; an objection greivous to a friend of humanity, and which, for my part, I have heard repeated so often that I would not be astonished if it presents itself at this very moment to the mind of the reader. The objection is this. The organic affections of the heart are now, and will always be beyond the resources of medicine. What then is the advantage of this nicety of precision, and of analysis in incurable diseases? What then is the good of ascertaining so perfectly the diagnosis of that which you will always fail to cure? What is the advantage of your paliative therapeutics, and your knowledge of the anatomical seat, or even the nature and degree of the lesion?

If all the diseases of the heart offered us these grave deformations of the valves, or of the orifices, which we have characterized by the double souffle, the vibratory tremor, the anasarca, &c., I would not endeavor to rebut the whole range of this objection. Indeed, without doubt, in cases of this nature, a local diagnosis is of small importance, when, on the one hand the general diagnosis, if I may so call it, is so easy; when on the other hand, the cure is so evidently impossible. But how true is it that all of these cases of disease of the heart are identical, under this double relation! Have we not just seen, in relation to pericarditis, of how many difficulties its diagnosis was sometimes susceptible? But will it be said of pericarditis, that it is one of the affections, in which the physician can shelter his defective diagnosis, behind the incurability of the disease? But treat only of the heart properly so called, though pericarditis associates itself without doubt, very intimately with its history; is it not only



in a differential point of view that to all the lesions of this organ, the terrible decree of Corvisart "*Hæret lateri lethalis arundo*," applies itself equally and without appeal? Has any one forgotten the sanguineous concretions in relation to which there was questions in our last observations? It is true that in almost all of these cases we have seen them followed by a fatal result. But can any one think that it will be always so? Does any one think that a proper course of medicine will never overcome these kinds of foreign bodies?—This would be a serious error against which I can appeal to our twentieth observation. Finally this practical fact is of so much importance that I cannot hesitate supporting it here by two observations which appear definitive to me, and which will complete the clinical history of this morbid production.

**OBSERVATION 22.**—*Temporary clots of blood in a patient attacked with an organic affection of the heart, and with emphysema.*—An old man aged 61 years, of a strong constitution but enfeebled by age, was brought to the hospital upon a litter, on the 17th of May, 1840. Having suffered for some days, and especially at this time, with a decided dyspnœa, the patient answered my questions with great difficulty, and in a broken voice. He said that he had always been very healthy. He complained only of a little weakness in his legs during the preceding year, and some dizziness for the past months. In going up stairs he also felt a little oppression and some palpitations. Since three or four days, this dyspnœa has so much increased as to decide him to come to the hospital.

*Present condition.*—His face was rather pale, his lips bluish, as were also his hands, which were cold; the jugulars were dilated, especially during expiration; there was no œdema of the lower limbs.

His inspirations rapid, not deep, dyspnœic, were 36 to 40 per minute. The resonance is quite good over the whole chest, but on both sides of the vertebral column there may be remarked a little prominence and the respiration is accompanied before, and especially behind, by a mixture of sibilant and even of disseminated subcrepitant rale.

The pulse is 89 per minute, irregular, intermittent, at one time small, at another well developed; there is no precordial prominence. The dullness is  $4\frac{3}{4}$  inches in both directions.—The point of the heart is felt in the 5th intercostal space but obscurely. Its beats are of medium force. Its sounds, from

time to time, deep seated, quite dull intermittent, are nevertheless accompanied, almost all the time by a double souffle, very distinct in all the precordial region, propagating itself even beyond that, effacing the two clackings in the region of the bicuspid and aortic orifices, continuing itself in the region of the aorta, even to the upper portion of the sternum, but at this point, permitting the second beat to be heard, which is dry, and a little like parchment. In the carotids, the souffle of the first time is scarcely heard. Nothing very remarkable in the other conditions. I prescribed bleeding of sixteen ounces, and warm applications to the lower limbs.

The following morning the state of the patient was almost the same. The pulsation of the heart as it was on the preceding day; the dyspnœa still considerable; the speech broken; the patient breathes only when half sitting up in bed.

M. Bouillaud orders two blisters on the posterior part of the chest.

On the following day the expression was diminished, the pulse was regular, it became fuller, and its intermissions were few and far between, it fell to 68-72, and the respiration to 20 per minute without any remarkable rale. The souffle covers only the first sound, it has lost much of its intensity. Finally he says he is well, and on the first of June demands his discharge.

I think it is useless for me to make any long comments on this observation. Who cannot see that in this patient we had all the signs of a medium hypertrophy of the heart with lesion of the left valves and especially of the bicuspid? It is not our purpose to insist on this point of the diagnosis; but what I assert, is, that independently of these fixed and permanent lesions, we here had clots of blood temporarily arrested in the cavities of the heart, and it is by them that I explain this extreme dyspnœa, the anxiety, the coldness of the extremities, the dilatation of the jugulars, and the intermittence and irregularity in the contractions of the heart, the pulse at one time small, and at another, more developed; it may be that the bronchial rales will justify us in the belief of the existence of an emphysema.

Such was my diagnosis; and especially so when the amelioration in the symptoms was so plainly visible, when I compared the souffle noted towards the end to that which I had verified at the beginning; this difference having appeared to me much too decided not to have some other cause than the

difference, comparatively so small, the 84 pulsations of the day of his entry, and the 72 of the day of his departure.

Finally, if this fact may strictly leave some doubts, it will not be thus with the following case, which appears to me protected from every objection in the point of view which now occupies us:

**OBSERVATION 23.**—*General hypertrophy of the heart, with thickening of the valves.—Triple sounds.—Clots of blood in the heart.—Pulmonary Œdema.*

Antoine Cambrowne, nineteen years of age, a cabinet maker by trade, was received into our service on the 15th of January, 1841.

This patient, of a delicate constitution and lymphatic temperament, said he was attacked two years since, with palpitations, with dyspnœa, upon ascending an elevation, phenomena preceded, and, to all appearances, caused, as the patient himself said, by a general acute articular rheumatism, which had existed for two months. The following are the practical symptoms which I noticed on his entry:

No swelling of the face nor infiltration of the limbs.

The pulse at 80, of a medium fullness, with some inequalities or rare intermissions. Precordial prominence of two or three degrees of the cyrtometer. The point of the heart beats in the fifth intercostal space; and this beating extends as far as the lower portion of the sternum, where the finger feels some tolerably strong pulsations. The precordial dullness is about  $3\frac{1}{4}$  inches square. The impulsion is strong and pretty well extended. No vibratory tremor. In ausculting the precordial region, one is struck at first with the hardness of the two sounds which are dry and like parchment—very distinct, without a very decided inorbid sound; nevertheless the first sound is accompanied by a sort of roughness or friction, which somewhat resembles the soufflé, especially so in approaching the lower part of the sternum. No soufflé in the carotids; cough very rare, two sero-mucous expectorations. Resonance good throughout. The same is true of the respiration, which is nevertheless accompanied, partially, on the left and almost in all the height on the right, behind, by a moist rale with small bubbles, heard only at the end of respiration, and recalling in some degree, crepitant rale. On the next day M. Bouillaud noted, in his turn, the following phenomena. The pulse 64, tolerably full vibrating, regular, not double. The valvular sounds are rather dull, and at the same time, loud without distinct soufflé, but they divide themselves clearly into

three by the doubling of the second sound. This triple sound, well marked in all the precordial region and the adjacent parts, is most distinct in the submammary region. The same crepitation a little humid. The chest is a little prominent behind and very sonorous. Some glairy, semi-transparent expectorations without tinge of blood.

Up to this time nothing is plainer than the diagnosis of this disease. We have, without a doubt, general and very considerable hypertrophy. We have, moreover a fibro-cartilaginous thickening of the valves without any sensible contractions of the orifices, and in the lungs perhaps an inflammation of the bronchiæ with tendency to emphysema.

Such at least was the diagnosis made by M. Bouillaud who prescribed, consequently, a bleeding of ten ounces, and cupping on the back, and both sides to the same extent, some pectoral ptisans and a portion with tincture of digitalis.

We remark, in relation to this diagnosis, that M. Bouillaud, contrary to his custom, does not point out the left valves as exclusively affected. Was it because the rudeness of the first sound, noted towards the inferior portion of the sternum, induced him to suppose that the right heart would naturally partake of the disease? I do not know but I would be so much less surprised if soon after we see him diagnosticate clots in the right cavities especially. As for myself, I confess that the lesion of the right valves appeared to me to be here a very probable coincidence, and I was even tempted to think that this lesion was concerned in the production of the bronchial rale, which, in this hypothesis would be rather a sign of œdema resulting from a little hindrance of the cardio-pulmonary circulation, and would rather be a sign of œdema than a symptom of the bronchitis with emphysema admitted by M. Bouillaud.

As to the nature of the valvular lesion, the tone of the sounds, and the absence of the soufflé did not allow any one to mistake a considerable thickening of the laminæ, without deformity, vegetations, &c., and as M. Bouillaud said, without any considerable contractions of the orifices. I wish it to be observed in relation to this subject, that we here have the triple sound; this triple sound may exist without that bicuspid contraction which I have mentioned above.

I will add finally this last remark, which confirms those already made, viz: that the triple sound is perceptible only in the condition of a certain abatement of the pulse; absent at the entry of the patient with eighty pulsations, whilst

there then existed especially in the right cavities a sort of raspy friction in the first time; this triple sound displayed itself on the next morning, the pulse having fallen to 64, and the rude sound of the day before having cleared.

The phenomena noted the following days sustain this remark, at least as far as it concerns the rasping sound.

Thus on the morning of the 15th, the pulse is at 72, the triple sound continues, but with a hard and rude tone. These are the expressions of M. Bouillaud; the following days the pulse falls to 56, and there is no longer a question of this peculiarity, the triple sound being noted, on the contrary, as perfectly distinct. Finally, under the influence of cupping and bleeding on the back part of the chest, the crepitation disappeared completely; and on the 20th of January the patient felt well enough to remain out of bed without our permission during more than three hours of the afternoon.

Here commences that part of this observation which especially interest us, and in fact in consequence of this imprudence, the patient, who had had cold feet, was seized with considerable dyspnœa: and when I arrived to visit him that evening, I found him suffocating, his respiration anxious, 36 per minute, his face and body covered with a profuse perspiration. the pulse 128 and vibrating, the sounds of the heart distinct with rude souffle in the first time; the resonance is good over the entire surface of the chest, but I heard on both sides, even in front, a humid and abundant crepitation with a mixture of sibilant rale, behind and to the left, without pain in the side, the expectoration purely mucous. I prescribed immediately a bleeding of twelve ounces and mustard plasters to the feet.

On the morning of the 21st M. Bouillaud noticed the following.

The relief afforded by the bleeding was but momentary. The oppression and anxiety are very great this morning: sleeplessness; abundant sweat during the night; paleness: pulse 120, regular, sufficiently vibrating, and contrasting by moderate developement with the force of the beatings of the heart. The sounds of this organ are perceptibly more obscure and more concealed towards the right than towards the left. The first is a little rough but without any distinct souffle at this time. The triple sound no longer exists. Respiration is at 40. Crepitation in the whole chest with tendency to souffle behind, which is owing to the frequency and force of the respiratory movement.



The resonance is tolerably good throughout. Expectoration is glary. The clot of yesterday firm and glutinous and without buff. The serosity is somewhat turbid. Bleeding of eight ounces, cupping upon the chest of ten ounces.

The 22d; the patient is better; the dyspnœa and anxiety have disappeared; the pulse is at 84 and well developed; the sounds of the heart less forcibly struck and not so rude; the triple sound has not again reappeared; crepitation less abundant behind, none before; resonance pretty good; the clot firm, glutinous and covered with a thin buff; the rondells of the cupping glasses are united in a clot of medium resistance.

The 23d; Improvement continues; the pulse is at 76 to 80; return of the triple sound but not so marked as before; yet a little crepitation behind.

The 24th; Patient doing well; triple sound returned; pulse at 68.

The 29th; Being charged with the morning visit, I find our patient in a state of dyspnœa worse than any we had observed. Half setting up in bed, he tossed about, uttering groans and even cries; his face pale and covered with sweat, as was also his body; his lips blue, his eyes half closed, and his nostrils dilated. Respiration at 48. Crepitation has reappeared in the whole chest. The pulse is small, contracted at 128 to 132. The sounds of the heart were rudely struck, without notable souffle, owing, perhaps, to the difficulty of analyzing them, resulting from the excessive quickness. The triple sound has disappeared.

We were told that this crisis commenced the evening before, the patient having eaten an excess of food brought from without.

I prescribed a bleeding of eight ounces, and blisters to the interior surface of the thighs.

The 30th; Continuance of the condition of yesterday, which has continued without interruption. The pulse, almost imperceptible, is at 108 to 112. The beats of the heart strike the hands with force. Nothing new in regard to the sounds of the heart except that they are notably concealed. Respiration is still accelerated and very high. Before as well as at the back of the chest there is heard, throughout, the firm and humid crepitation before spoken of. The extremities are cold; the patient is restless: says he is suffocating.

Persuaded for my part that he is devoted to an inevitable death, I deplored the slowness of the approach of asphyxia; and not daring to return to blood letting, I ordered two blis-

ters to the posterior part of the thorax, mustard plasters to the feet, an infusion of Linden-flowers and a potion of æther.

The 31st; The symptoms are abated. Patient is stretched on his bed; the pulse is full, regular and at 68 to 72; the sounds of the heart are perfectly defined; they are no longer concealed, and in this respect they differ singularly from those of yesterday.

M. Bouillaud added then to his first diagnosis,—

Latterly *Capillary Bronchitis to the left and disseminated pneumonia. Probable concretion of the heart and especially in the right cavity.*

We will add at once to terminate this history, which has been rather long perhaps, but the interest of which will probably excuse the narration, that these crises of dyspnœa returned no more; but that first an erysipelas of the right arm and afterwards a rheumatismal swelling of the left knee, and of the right wrist kept the patient in our service until the 2d of the following May. During this stay I noticed many returns of the triple sounds spoken of; and I found, besides, on the 24th of February, a souffle of the first time in the inferior hollow in the sternum, ceasing to be hard when you passed that point. The 2d of April this sternal souffle is even accompanied by a distinct purring. Finally, the 2d of May the day of his discharge, there could yet be noticed a souffle of the first time in the right cavities; and some sounds, very dry and like parchment in the region of the left cavities.

What need is there of commenting on this observation? Can not any one tell that it presents to us a perfect history of concretions in the heart.

And finally, is not his interest assuredly very small, who does not see, after this fact, that if clots of blood can be deposited in the heart before death, it is not less true that, with suitable treatment, they can be removed or absorbed, and often very quickly? Of what great importance then is this point of diagnosis to the practitioner? Let us remark also that in the point of view of the symptomatological study of lesions of the right side of the heart, this fact is to be added to those we have pointed out above, in which the morbid sounds were heard, especially towards the lower hollow of the sternum.

Let us also note the gradations presented in our patient in relation to the constancy and tone by the morbid sound so well localized and perhaps responding, according to my notion, to the orifice of the pulmonary artery, increasing gradation

with him, and to which the sanguineous concretion could not have being foreign.

I will finally conclude with a last remark. It is, that I confess it is difficult for me to explain, with M. Bouillaud, the humid and general crepitation, so ready to reappear at each of the crises ; so ready also to disappear with them, by a disseminated pneumonia and even by a capillary bronchitis, an affection always so tenacious and often so rebellious against the most energetic medications.

The characteristics of the expectoration, the absence of dullness and some previous observations, analogous and verified by autopsy, induced me to view it rather as a case of acute pulmonary œdema, a disease better known, and more observed in general under a slow and chronic form, but, in my opinion, not the less real ; at other times very rapid in its progress, compromising, in some instances, the existence of the patient, but also having this advantage ; that connected sometimes, as in the present case, with a stoppage, sudden and momentary, of the venous circulation, it will disappear very quickly if the obstacle itself is removed. This and the preceding observation demonstrate, then, the great practical importance of knowing how to diagnosticate, and how to separately oppose, when necessary, these sanguineous clots, an epiphanomenon grave and, it may be said, very frequent in the organic diseases of the heart. But how much more apparent does the importance of this become when we reflect that these morbid productions are not less possible in a large number of other diseases, such as pneumonia, pleurisy, rheumatism, &c. ; diseases in which a daily examination of the heart can only, very often, give us the key of certain febrile conditions which seem to survive their original cause and in which every one has so often seen an astonished examiner recur, for want of a more exact diagnosis, to the idea, so often chimerical, of a diathesis.

Again, how much better will this necessity of an exact appreciation of what passes in the heart, be demonstrated to us if I add that in all of these diseases, frequently, in the place of sanguineous concretions, that is to say of disorders with very decided physical signs, we meet, as a complication of them, a beginning endocarditis ; the primary origin of so many incurable lesions, and which then only reveals itself to us by signs which a practiced ear alone can catch ; this disease being, I may say, as difficult to diagnosticate at its commencement as it is important to oppose at this period of its devel-

opment. I have at hand very numerous observations corroborating this coincidence between a disease of the heart and a phlegmasia. I have even among other facts, not only in my recollection but under my eyes at this moment, a case, the only one, it is true, of a typhoid fever, in the course of which an organic souffle announced to us the very unexpected complication of a disease of the heart. I cannot relate all of these cases, but will content myself with merely mentioning them. There is one, however, which I will take as an example, because it will offer this curious peculiarity which we will then perceive without a possibility of a doubt, a disease of the heart commencing under our eyes, and very soon, thanks to the active treatment suggested by its diagnosis, to disappear in some way without leaving any traces of its existence.

*OBSERVATION 24.—Acute articular rheumatism; rasping souffle at the bicuspid orifice soon after the entry of the patient. Chlorotic souffle at his departure.*

Edward Papler, a hair-dresser, aged twenty three years, was received into our service on the 25th of May, 1841.

This young man of an average constitution, of a lymphatico-sanguineous temperament, was attacked one year since by an articular rheumatism, general and febrile, which deprived him of all movement during seven or eight days, and lasted six weeks.

Treated at the "la Pitie" hospital by bleeding, laudanum, cataplasms and opium pills, he was discharged relieved, but some pains still remaining in the fingers and toes. He remained besides subject since then to feeling vague pains, and experiencing palpitations whenever the weather changed.—Six days before, without any known cause, he was attacked by pains in the two wrists, which quickly localized themselves in the right wrist; accompanied by a little chillness and febrile heat. The following was the state in which we saw him:

The right wrist as well as the hand was of a diffused redness and of a temperature manifestly higher than that of the opposite side. It presented an evident tumefaction, expressed, in comparison with the left wrist, by a difference of  $2\frac{1}{2}$  lines. The subcutaneous veins on the diseased side were more prominent than those of the sound. The pain was almost continued, very acute and increasing on the least movement. The face generally quite pale and meagre as is also the whole body. Nothing remarkable in the digestive or respiratory passages. The heat of the trunk is moderate and a little moist. The

pulse is at 96–100, full and vibrating, especially in the right radial where it is preceptibly more developed than in the left. No precordial prominence. No displacement of the point of the heart—no abnormal dullness. The impulsion is very distinct, but moderately extended and without vibrating tremor. Finally, the sounds of the heart are regular, well struck and very distinct, *without souffle*—no bruit de diable in the carotids. I prescribed a bleeding of 12 ounces.

In the morning (26th) the patient feels himself relieved; he had slept better; the hand and the wrist are less red, and rather less swollen; there might be remarked a slight roseolar eruption towards the middle of the posterior face of the radio-carpal articulation. The veins are still developed; the pulse is at 84, tense full, resisting, and, like it was yesterday, evidently fuller in the right than the left; the precordial dullness is perfectly normal; but in the region of the left cavities there was heard this morning for the first time a well marked souffle, concealing almost entirely the valvular clacking a little rude, rather rasping, perfectly distinct towards the left auriculo-ventricular orifice, then diminishing in intensity as the sternum was approached and even disappearing almost entirely towards the summit of the chest, and then being replaced by a very distinct clacking sound. The clacking of the second time is, on the contrary, without souffle and distinct. No trace of souffle in the carotids or subclavians, in which there was heard, by propagation, a double clacking, the first sound of which is without notable souffle. No precordial pain. The clot of yesterday showed a very thin buff. The serosity is rather turbid.

Let us stop here some moments. The results of this second examination were, relatively to the condition of the heart, so different from those of the day before, that M. Bouillaud desired very much, before dictating that which precedes, that I should auscultate in my turn; and hardly had I applied my ear, when I heard and announced, in fact, a souffle of the first time, such as had been described. I was even so much struck with its rudeness, that not being able to think that it dated only from the preceding night, I attributed it immediately to the attack of rheumatism which supervened one year before, and since which the patient had still some palpitations, presuming that this souffle had apparently escaped me the evening before, I even avowed this supposition to M. Bouillaud, who repeated it in his public lecture in terms too flattering for me to mention here. And, notwithstanding this supposition, I only stopped myself with reluc-



tance from denying the existence of a souffle, the existence of which so many reasons had induced me, in advance, to admit and almost positively to assert, when I thought of the minute care which I had used the day before, having decided only after prolonged examination, and after being well convinced of its absence. Finally we shall now see what will happen from this souffle.

M. Bouillaud, wishing suitably to oppose both the actual local manifestation of rheumatism, and the internal inflammation shown by the fullness and vibration of the pulse, and the state of the heart itself, recent, perhaps, and indicated by the souffle, prescribed a bleeding of 12 ounces, and cupping on the wrist and hand of 8 to 12 ounces.

The 27th, the patient was better ; he moved his hand much more easily, and the swelling and redness of it were again diminished. He, however, felt some new pains towards the shoulders, in the right arm and in the left fingers. Besides this, the heat of the skin was very moderate ; the pulse, less tense, less prominent than the day before, had fallen to 72-76. Auscultation often tried, could hardly distinguish, at the end of the first time, any vestige of the rasping souffle, which, the day before, entirely concealed the clacking. There was heard on the contrary the first sound well struck, and the change since yesterday, to use the words of M. Bouillaud, could not have been greater. There was no souffle in the carotids or subclavian arteries. The clot is very much retracted, with the buff coat a little cupped up, and of medium thickness. The rondelles are united in a mass tolerably glutinous, and the serosity is not red.

Could any one doubt even that the souffle of the preceding day was not really a souffle of new formation, and the positive index either of a beginning endocarditis, having already thickened the bicuspid valve in a manner to impede its proper action ; or of some fibrinous concretions deposited upon the laminæ, and producing the same result ? No ! doubtless none but a morbid sound of very recent date could disappear in so short a time ; but this success was also a powerful motive for following and completing the cure even at the risk of impoverishing momentarily the mass of the blood. M. Bouillaud then did not hesitate to order a new bleeding of 12 ounces, induced moreover, besides the important indication I have mentioned, by these new pains, vague as yet, but which could well show that the rheumatic flame was not yet extinguished.

And, in fact, that which proved to us that this second indication was as real as the first, was that on the 28th, the pains of the left shoulder were more acute than on the previous day. There has supervened, moreover, a little soreness of the throat, with a slight swelling of the right amygdala, a redness of the pharyngeal membrane. The clot is very much retracted with thick and cupped buff. Nevertheless, the diseased hand and wrist have returned to a normal state. The pulse is at 68-72, and the first sound of the heart is even more distinct.

This time the only danger which existed was that of a return of the rheumatism, but even this was a sufficient motive for action. M. Bouillaud therefore ordered a final bleeding of 12 ounces.

The 29th. The patient is well ; there existed no pain except that of a slight sore throat. The pulse is at 68 ; the first sound is as well struck as the second, without the mixture of any distinct souffle. M. Bouillaud gave him some broth.

The 30th. Improvement continues ; the pulse is at 60 ; nothing new in relation to the heart.

The 31st. The pulse is at 60 ; there was noticed a very slight souffle, soft, chlorotic, and terminating the first sound, the clacking of which is very distinct. This souffle which extends in to the substernal aorta, coincides with a well marked bruit de diable in the left carotid. With this exception the patient does very well.

The 15th of June he demanded his discharge, being perfectly cured. There was yet heard a distinct bruit de diable on the left, in the sitting position only, and a slight souffle, very distinct but chlorotic at the first time of the heart towards the aortic orifice, whence it extended into the substernal aorta. There existed no sign of any organic lesion of the heart. Is it possible, I ask, to meet a fact more remarkable in every respect ? and was not each particular of it worthy of interest, even to this chlorotic souffle, supervening towards the end of the treatment as if to dispose of itself in favor of the organic nature of the rasping souffle which had preceded it, and to confound for once more those critics of whom I have spoken above, who assert that M. Bouillaud in his observations could not well know how to distinguish these two souffles from one another ?

I conclude ; that which I wish to be well observed in this case is, that instead of knowing how to make a precise diagnosis, M. Bouillaud has been able to cure an organic dis-

ease of the heart in its commencement ; that there are then amongst these grave lesions some cases in which our therapeutics are not purely palliative.

But without wishing to dwell here for an etiological discussion, which would take us too far from our subject, let us examine our twenty-three preceding observations on the diseases of the heart, in reference to the probable cause ; and what do we find ? Seven times this cause was unknown, twice it was traumatic, five times a catarrh or an acute bronchitis, and nine times a rheumatism. Here then are 16 out of 23 where every thing induces us to believe that an endocarditis, which doubtless passed unnoticed, was the starting point of these sad diseases becoming at a later date the opprobrium of our art, which when commencing might have been arrested if we had known how, first to diagnose, and then to combat them.

These practical considerations respond victoriously to the objection which we contradict, and are sufficient to sanction all that our diagnostic studies can seem to offer of scientific precision, and, I will say, almost of exaggerated pretensions. Who in fact does not understand the usefulness or, I should rather say, the necessity, in cases such as I have analyzed, of the habit of these special explorations, of these minute analyses in the most complex cases, of which we have shown the means and the result ?

But I now say more ; in exercising ourselves in this kind of diagnosis of the diseases of the heart, whatever may often be the symptomatological obscurity in which they envelop themselves, in studying to discover these affections whenever they are present, we prepare ourselves even by this, to know how to determine with certainty the cases in which they are not, whatever may be the functional disorders which seem to announce formally to us their existence ; and in how many cases does this negative diagnosis require as much experience as the affirmative ! It may be divined that in this place I wish to speak of nervous palpitations, and in particular of chlorosis, a disease insidious in its symptomatological manifestations, and which, under different forms, causes inexperienced practitioners to imagine it one of the affections of the centre of circulation. Shall I mention in relation to this, one of our most distinguished apothecaries of Paris, who, believing himself, from the opinion of his physician, affected with an organic lesion of the heart, referred to this cause, very frequent palpitations, pains in the precordial region, suffocating sensations, &c., and in whom all these symptoms have disap-

peared two years ago without returning, because I persuaded him to oppose them only by means of antispasmodics, or rather because I told him, and I have been happy enough to make him believe, that his organic lesion was purely illusionary? Shall I mention one of our chlorotics, received into our service and come to Paris to be cured of an hypertrophy of the heart which did not exist, and against which the principal physicians of one of our northern cities had expended all the resources of antiphlogistic medication, and all the pharmaceutic combinations of digitalis?

I cannot tell with how many similar facts I might support this demonstration. I do not exaggerate in affirming that, in the two years of my practice, not a week passed without our receiving into our service many of the unhappy victims of the error in diagnosis of which we speak. Shall I say also, that if simple cases of chlorosis were very often sent to us as diseases of the heart, sometimes, also, in consequence of an inverse mistake, I have seen, what is more astonishing, real organic diseases of the heart, very grave and already far advanced, sent to us for chlorosis?

And it is when like errors happen every day; when some gave iron in cases which need bleeding and digitalis; when others bleed, when, on the contrary, the blood should be strengthened, that they ask us what is the advantage of our precise studies, and our minute researches upon questions so teeming with mistakes not less shameful to the physician than dangerous to the patient! And it is this very class of people, who err in this way, who reproach us with wishing to throw an excess of light, a superfluous glare, upon the path which they so blindly tread.

Finally, I believe I have said enough to reduce their objection to its proper value. I believe that I have sufficiently demonstrated, that, if in presence of certain organic lesions of the heart we are all now, and ever will be, it is true, equally powerless, it is not less incontestible that, with the exception, of these sad circumstances, there are yet some therapeutic palms to cull—more of practical success to attain; and that this part is large enough to repay us worthily, the pains and efforts it will cost.

Happy am I in my own estimation, if I have thrown any light, in this memoir, upon any clinical questions; and especially if I have stimulated the zeal of our young practitioners, for the diagnostic studies which constitute, in my mind, one of the most beautiful parts of our art.





